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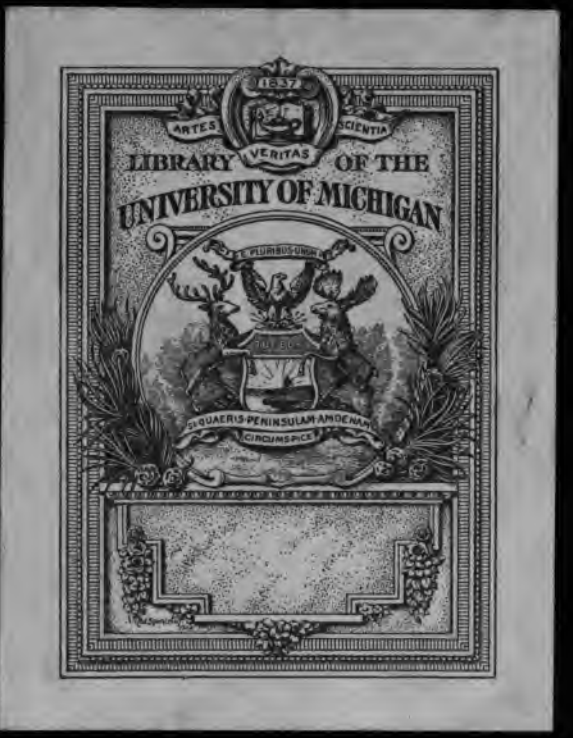
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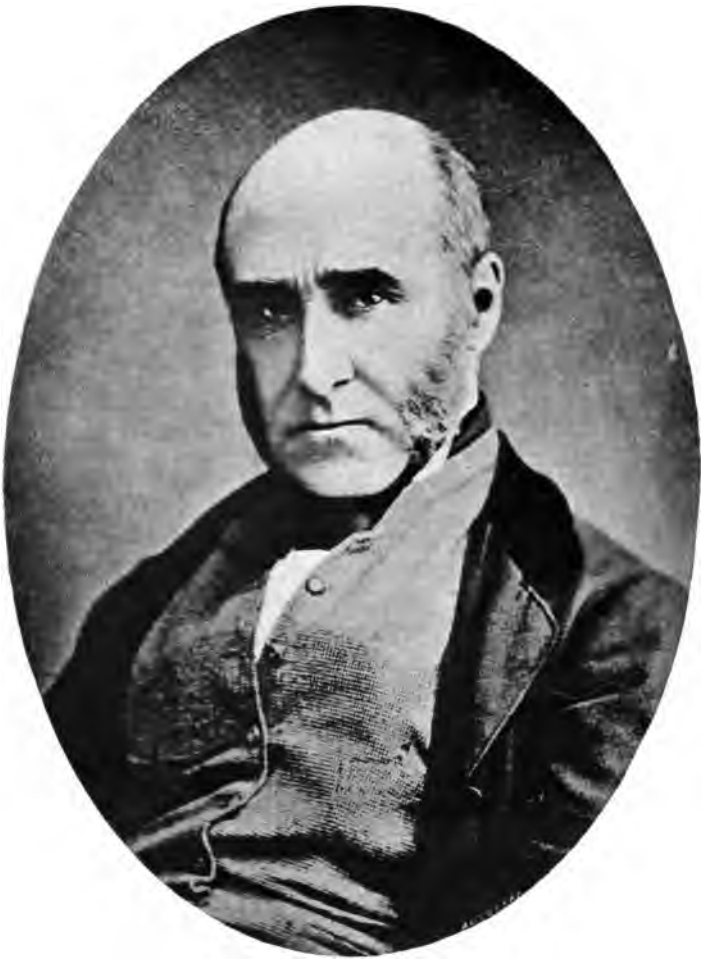

SIR JOHN SIMON, K.C.B., F.R.S.

(*From a Portrait taken in* 1876.)

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(Law III, 8.)

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1894. Cameron, Spottiswoode, M.D., Municipal Buildings, Leeds.
1902. Clayton, F. H. A., M.D., H.M.S. "Prometheus," c/o The Admiralty, S.W.
1889. Cooper, C. H., A.M.I.C.E., Local Board Offices, Wimbledon.
1891. Davies, D. S., M.D., Sanitary Offices, Bristol.
1902 Elkington, J. S. C., M.D., Office of Chief Medical Officer, Hobart, Tasmania.
1894. Evans, W. Arnold, M.D., Town Hall, Bradford.
1897. Fraser, A. M., M.D., Town Hall, Portsmouth.
1903. Hendley, A. G., Major, I.M.S., c/o Grindlay & Co., Bombay.
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1888. Wilson, J. Mitchell, M.D., County Hall, Beverley, Yorks.

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1888. Bately, J., M.D., High Street, Gorleston, Great Yarmouth.
1895. Borthwick, T., M.D., Kensington, Adelaide, South Australia.
1883. Bovill, Surg. Lieut.-Col. Edward, M.D., C.M., I.M.S., Bengal, India (*retired*).

Elected

1889. Bruce, David, M.B., Lieut.-Col. R.A.M.C., c/o War Office.
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1885. Crawford, D. G., Surg.-Major I.M.S., Mymensingh, Lower Bengal.
1891. Crook, H. E., M.D., 23, Dalby Square, Cliftonville, Margate.
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Elected

1885. Tomes, Arthur, Surg. Lieut.-Col. Ind. Med. Service, St. Leonard's, Malvern.
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TRANSACTIONS

OF

The Epidemiological Society of London.

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SOME REMARKS ON THE EPIDEMIOLOGY OF PLAGUE.

By W. J. SIMPSON, M.D., F.R.C.P.

(*Read: November 20th*, 1903.)

HAVING consented to read a paper on Plague, it occurred to me that the occasion would be a fitting one for obtaining the benefit of the opinion of the Society on several questions connected with the epidemiology of Plague, which are somewhat obscure, and which are deserving of discussion.

To begin with: there is the comparatively rapid retrogression of Plague from Western Europe in the latter part of the seventeenth century, and its disappearance from Eastern Europe in the middle of the nineteenth century. To the epidemiologist they are important facts not explained by the Fire of London or by any other local cause such as appeals to the sanitarian, but are evidently dependent on wider influences which it is desirable to investigate. True, there were no more epidemics in London after the Great Fire; but that was more or less a coincidence, just as at Delft, in 1537, the town was decimated with Plague after a great fire in 1536.

It seems to me that possibly some of the causes of the retrogression may be ascertained by studying the mode of advance of the present pandemic of Plague.

It will be remembered that Plague has been endemic in the Province of Yunnan for many years, but it was only in 1894 that it overflowed its boundaries to such an extent as to reach Canton and Hong Kong, two of the most important commercial emporia in the Far East. Admirably situated for commerce, Canton and Hong Kong are the great marts and distributing ports for the produce of Southern China; and Canton sends its merchandise down by the Canton River on large and small native craft to

Hong Kong, where it is stored in large warehouses until ships arrive to take it away in exchange for the cargoes they bring. Hong Kong, though not more than sixty years old, possesses one of the busiest harbours in the world, and has trade connections, not only with the southern ports of China and the neighbouring islands of the Pacific and Chinese Seas, but also with India, Australia, Japan and America.

It was from Canton and the Province of Kwantung that Hong Kong became infected with Plague in 1894, and which continues periodically to receive fresh infection. Plague since 1896 has also become endemic in Hong Kong. A great commercial centre, continually exposed to fresh infection and in which Plague is endemic, is apt to be dangerous to places with which it is in frequent communication, and it has thus happened that Hong Kong, besides being a great distributor of merchandise, has become also an active centre for the distribution of Plague. Sea-going ships have conveyed the infection over the seas to India, Australia, Japan and America, and coasting steamers have distributed it to adjacent ports. It has not always been possible to indicate the ship that conveyed the infection or the exact agent by which the infection was carried, whether by man, by animals, by infected clothing, or by infected merchandise; but it is possible to exclude the infection being conveyed in any other way to Japan, Australia, and America, and there is sufficient evidence to leave no doubt as to the infection being carried by shipping having commercial relations with infected ports in Southern China.

The history of the progress of Plague since 1894 is that few maritime countries participating in the rivalry of the commerce of the East have not been more or less exposed to Plague by the arrival of ships from infected ports.

Whether from precautions taken, or from conditions in the port unfavourable to the lodgement of Plague, or from the rarity with which ships carry infection on them or from ships being generally unfavourable to the propagation of Plague, the majority of the ports subjected to the risk have escaped. But though there has not been general infection of the ports, yet the infection has been carried to many parts of the world.

They include ports in Asia, North and South America, Australia, Africa, and Europe. It is a distribution, as far as is known, dissimilar to any former spread of Plague. Its main feature is that it has followed a maritime course.

Hong Kong is separated from most places by the sea, and the infection has not passed overland through China, but has followed the trade routes of the sea.

It is a very remarkable fact that Plague has not spread very far inland in China, and that hitherto its chief ravages have been limited comparatively more or less to that portion of the country near the coast. The infection on land has followed chiefly the routes of busiest intercourse. There are no railways in Southern China, so that the extension and tendency to extension which has been observed in India and South Africa along the lines of railway has been wanting, and the disease has made no extensive inroads into the interior of the country, except in those districts in which there are waterways connecting them with infected localities. In the case of the infected provinces of Kwangsi and Kwantung, and Fokien, a range of hills which forms a natural boundary between the north and the south, which restricts commercial activity between them, also prevents Plague from passing northwards. But apart from this natural obstruction, it seems to be a characteristic of Plague not to spread much beyond the towns and villages on the more frequented roads of trade.

The main epidemiological facts connected with this pandemic of Plague which we are now interested in are firstly, that the endemic centre from which the disease sprang is far away from the other known centres: a circumstance which, with the facilities for better information now obtainable, allows of a clearer view of the spread of Plague; secondly, that the disease has not passed overland in China except by well-known and busy trade routes; thirdly, that it has followed the maritime trade routes; and finally, that the distribution is dissimilar from any previous one.

These facts may be applied to a consideration of the questions previously stated. The connection of Plague with the modern trade routes may help to explain the decline of Plague in the latter part of the seventeenth century.

If we turn our attention to the history of the commerce of European nations, we find that the great centres of activity shifted from time to time. The nations in possession of the trade of the East have been the richest.

From the tenth to the sixteenth century the Venetians possessed almost a monopoly in the commerce between the East and the West, their only rivals being other Italian States, such as Genoa and Florence. Italy, more particularly Venice, was practically the gateway through

which the produce from India, China, and Persia passed into Europe. The merchandise was brought overland in caravans to the shores of the Mediterranean or Black Sea, and thence by ships to the Italian State, which was the great distributing centre for Europe. On their way through Mesopotamia and neighbouring countries the caravans passed through endemic areas of Plague. The Venetians, with their opportunities, were the first to learn that there was a connection between trade passing through countries affected with Plague and the conveyance of that disease to healthy localities, and for self-protection they were the first to practise against ships preventive measures in the form of quarantine, which was based on the medical knowledge of the day as regards the duration of infection. The great trade routes from Venice to the north-west of Europe, to the Baltic, and to the North Sea, were not by sea but by land through central Germany. The Hanseatic League, that great confederacy of towns for the furtherance and protection of trade on the north, was the connecting link between Venice and the north. It carried on an immense trade with Venice. The great commercial cities of Bremen Danzig, Lübeck, Hamburg, Cracow, Ratisbon, Augsburg, Nuremberg, Frankfort, and other towns were connected by these land routes; and periodical fairs were held in them to which merchants flocked from all parts, bringing their goods and exchanging them for others. They afforded facilities if Plague was present for its extension. During this period Plague appears to have prevailed periodically and with great persistence in Europe.

The two great discoveries at the end of the fifteenth century and the beginning of the sixteenth, viz., the discovery of America and the discovery of the Cape Route to the East, were gradually to effect a great change in commerce, one of which was the transformation of a commerce which was exclusively overland or coasting to a sea commerce, and the other was to change the routes of international commerce so as to deprive the Italian States of the monopoly which they had possessed for several centuries. Venice, the principal mart of the products of the Orient, was not long in discovering the injurious effect likely to arise from the discovery of the Portuguese, and in the sixteenth century its Government made advances to the Portuguese with the object of buying everything brought by the Portuguese from the East. These proposals were rejected. Trade, however, seldom becomes suddenly diverted from its customary routes, and though much of

the commerce of the East was shifted to Lisbon, a great deal remained in the Mediterranean shared by Italy and France; and it was only at the beginning of the seventeenth century, when Venice lost its power in the Levant, and when the Netherlands and England began to take the place of Portugal and Spain, by which the commercial activities of Europe with the East were transferred to the ports in the North Sea, that the roads northwards from Venice and Marseilles became no longer the routes by which the produce of the East was carried to northern towns. The Hanseatic League came to an end about 1641. The thirty years' war, from 1618 to 1648, which had been the means of spreading Plague largely in Europe, had practically destroyed the mercantile intercommunications between the North and the South, and at the same time had destroyed the League.

France, which was the only other country having direct dealings with the Levant through Marseilles, was in a state of misery and disorganisation during the first half of the seventeenth century, and was afflicted not only with Plague, but also with famine. Its commerce with the Levant and North Africa was brought to its lowest point, on account of piracy in the Mediterranean and the unfriendly attitude of the Turk.

A new route from Persia by the Caspian Sea, Astrakhan, Novgorod and Narva, was accordingly opened out in 1630, by which for a considerable time the produce of Persia and the East was conveyed to France.

The abandonment of the Mediterranean as the centre of commerce for Europe, the shutting up of the Levant as the high road for the conveyance of the produce of the East to the West, and the transfer of commercial activity to Amsterdam and London, whose connections with the East were by sea and not by land, and, consequently, the avoidance of the former intimate connection with endemic centres, were changes which came into operation in the early part of the seventeenth century; and it appears that it is in these great changes in the commercial relations of Western Central Europe that the chief factors in the rapid disappearance of Plague from Europe are to be sought, once the influence of war in Central Europe and famine in France were over, rather than in any great social change effected at that period.

Under these circumstances, quarantine, as practised in the Mediterranean ports, became easier in its application and more effective in its results. Plague continued at

intervals in the neighbourhood of the new overland route, and in those countries with which Turkey was at war, for more than another century, but it spread very little out of the beaten track.

The decadence of the trade of Bagdad, and other towns on the Euphrates and Tigris, with the appearance of steamers at Suez in 1839, bringing merchandise from India to Egypt, instead of much of it *viâ* Persia and Syria, seems to account also for the retrocession of Plague even from Turkey and Egypt in the middle of the nineteenth century.

If these considerations are correct, the risk of the present pandemic gaining a firm hold in Europe is at present not nearly so great as in former times. The ships coming from infected areas are cleaner, and not coasting vessels. Infected ports are notified, and thus precautions based on modern preventive methods can be promptly applied to ships coming from them; while the machinery for detecting and dealing with early cases, where the disease evades the sanitary authority and is imported, is more powerful and efficacious. These are factors all in favour of the prevention of the spread of Plague. On the other hand, our knowledge of the methods of the extension of the disease, once it has gained a footing, is very limited, and, as a consequence of this limitation, there is always danger. One of the most surprising facts as regards the existing prevalence of Plague is the small amount of money and time which has been devoted to experiment and research on this important question. Except at the stage of alarm in 1897, systematic research has not been much in evidence, and, accordingly, we are not much wiser than before, as to the best means of preparing for defence against a serious attack of Plague. I think too much confidence is placed on the sanitary condition of Europe, and on the assumed immunity of Europeans against Plague.

Apart from this, there are several questions that arise in one's mind: Will the sea, with all its advantages, permanently hold its present position in trade? Will the old overland trade routes never be made use of again? Already there are projects for railways through Asia Minor, and it is unlikely that the rich and fertile regions of the Euphrates will continue to be neglected. There will also be in the future more direct communication with Æthiopia; and already the great trans-continental railway of Russia and its other railroads tap regions which have been suspected to be endemic centres of Plague. In the development of commerce of the twentieth century, the Mediterranean may

assume again a commanding position; and, unless the endemic centres are dealt with at their source, the risk of Plague becoming once more a European disease will be considerably enhanced.

The next point is the seasonal limitations of Plague. There may be in any locality a few cases of Plague all the year round; but it is only at a certain season that the disease becomes epidemic. This season may vary somewhat in different localities, but it is nearly always the same in the same locality. In Northern Europe, the usual season is late summer and autumn. In Asia Minor, it is generally in spring and summer. In Egypt, it is in the spring and early summer, thus becoming earlier, as it were, the further south it occurs. In India there may be two epidemic seasons: one in January, February, March and April, which is the most common; and another in August, September, October and November. In countries south of the Equator, Plague becomes epidemic at a different time of the year to Europe, but always in the corresponding season. Cape Town and Sydney have their epidemic season in March, April and May. When the season of the year for Plague epidemics in several localities is studied, it is evident that the epidemics are governed by some periodical law. If Plague recurs in an epidemic form in a given locality, it is almost invariably at the same season of the year as the preceding epidemic.

The duration of an epidemic is also very much the same everywhere. Once established, it runs a certain course, whether met with by preventive measures or not. The latter may mitigate its effect, and keep it under control; but they have little influence on its duration. A comparison of the charts of epidemics in different places shows them to be characterised by a similarity in type. First of all, there is a period of hesitancy more or less prolonged; then there is a sudden but fluctuating rise, which reaches its highest in about the course of three months; then there is a fluctuating fall, which is much more rapid than its ascent; and, finally, the disease lingers in a sporadic form for some months.

There is a great similarity in the behaviour of different epidemics. Mean temperatures above 83 deg. Fahr., and mean temperatures below 50 deg. Fahr., seem to be inimical to Plague epidemics. There may be a small outburst in winter, such as occurred in Vetlianka; but these are very exceptional, and are more usually of the pneumonic type. In the Hong Kong epidemic any continuous temperature

above 83 deg. Fahr. was followed by a decline of the epidemic, which did not begin again until the following spring. In the Bombay epidemic there was always a fall when the mean temperature was above 82 deg. Fahr., and sometimes when it reached 80 deg. Fahr. In Cape Colony and Sydney there was a decline when the temperature reached a mean of 50 deg. Fahr. In the Cape it was observed that ten days to a fortnight after a rise in the mean temperature there was a rise in the number of cases of Plague. No very marked influence seems to be exerted either way by slight rains. If anything, slight rain with heat appears to favour Plague; whereas heavy and continuous rain—especially if the cause of floods—seems to be unfavourable to Plague prevalence.

Why Plague is favoured strongly by seasonal conditions is one of the problems still to be solved. Season, with its meteorological factors, is a composite force, and as such operates in more than one way on the agents and media connected with Plague. For instance, it affects man's constitution in various ways through its influence on the air, soil, and food which react on man; it affects the Plague bacillus in a similar fashion, and it affects animal and insect life as well. The difficulty lies in differentiating the main factors of which season is composed, and in determining the exact influence of each on man, the plague germ, and on animals and insects concerned in the spread of Plague.

That a mean temperature of 83 deg. Fahr. should exert so marked a control over an epidemic of Plague, while the Plague bacillus flourishes in man at 98 deg. Fahr., and in birds at 107 deg. Fahr., is perplexing, and leads one to suppose that the influence is not a direct one on the Plague microbe itself, which seems to be able to develop at considerably higher temperatures than 83 deg. Fahr. Especially is this view emphasised when it is considered how much the infection is a house infection, where direct sunshine plays a very unimportant part, the microbe being never exposed to any very high aerial temperature, or to any exceptionally low temperature which might destroy it. Connected with this circumstance is also the peculiar fact that on the decline of an epidemic, infected articles and houses in the infected locality appear to lose their power of infection for the time being, until the favourable season comes round again. It is well known that in the Plagues of London, Marseilles, Naples and Egypt, that towards the end of the epidemic the inhabitants who fled when the epidemic was increasing have flocked back to infected houses, slept in

infected beds, and worn clothes of those who have died of Plague; and yet, beyond a number of accidents here and there, that general infection which was to be feared has not taken place; and yet when a recrudescence takes place in the season of the following or a subsequent year, the infection is frequently—in Hong Kong it was largely—connected with Plague in the same house in the previous year, or in the year previous to that. When infected articles are transported to a new locality, and reach it at the commencement of the season corresponding to that which favoured the epidemic in the first locality, there is risk of the disease spreading, while the opposite phenomenon occurs when the infection is introduced at the wrong season. It would appear that there is then little or no risk of extension, for that year at least. We have an example of this in Marseilles at the present time. Measures taken against an infection introduced in early autumn, even though the disease has extended somewhat, are, if this is correct, more associated with successes than under similar conditions in spring.

The only research with the object of investigating the climatic factors in Plague epidemics is that of Mr. Baldwin Latham. He is not a medical man. Though we may demur to the interpretation which he gives to his observations, yet his facts are none the less of the greatest value. He has shown that when the air is warmer than the earth condensation takes place, but when the earth is warmer than the air evaporation takes place; in other words, that temperature exercises an enormous power in the elimination of vapour from the ground. By a series of observations he has further determined the periods of the year when exhalations escape from the ground, and the quantity that then arises, and he finds that the greatest exhalations correspond very closely to Plague prevalence. He deduces from this that a high temperature of the air, by raising the temperature of the dew point, and causing condensation to take place, stops Plague; while a fall in temperature means a fall in temperature of the dew point, and the tensional difference between a low dew point and a high ground temperature would at once lead to exhalations which, Mr. Baldwin Latham thinks, would lead to the liberation of Plague bacilli from the ground. Here I think we may join issue with him. It sounds like the old supposed cause of malaria. It is not improbable that the vapours favour a condition of susceptibility of the organism whether of man or of the lower animals, but it is unlikely to go

further. The hygrometric condition of the soil and earth temperature may possibly affect the development of the Plague bacillus, or some carrier of the Plague bacillus, not necessarily to man, but to and between the lower animals, and which mostly find their habitat in the nests of these animals. In this connection I may remark in passing that the phenomena observed within the last month of strong electrical currents in the earth disorganising the telegraph services, indicates that the conditions of the soil are influenced considerably by meteorological changes; and secondly, that the conditions of the soil may possess powerful properties.

It is a very remarkable fact that nearly throughout the year Plague rats have been found in Port Elizabeth, and yet it is only on occasion man becomes affected. Similarly in Hong Kong a few Plague rats are to be found during the winter, but it is only in the spring and summer that the Plague becomes epidemic amongst them and the mortality rises. This mortality precedes the mortality in man. What is it that limits the spread of the Plague among rats in the autumn and winter in Hong Kong and in corresponding seasons in other places, and what is it that spreads the disease among rats at a season which immediately precedes the Plague season in man?

Plague bacilli were discovered in fleas by Ogata in 1896.

During the epidemic in Cape Town, fleas were found to contain Plague bacilli by Dr. Gregory, and recently Plague bacilli have been found by Dr. Hunter in bugs in Hong Kong. Has the life-history of the fleas belonging to the rat any corresponding connection with the prevalence of Plague in the rat? Is the season of dormancy, egg, larva and pupa stage, synchronous more or less with the season of lowest prevalence of Plague in rats and *vice versâ*? Is the season of highest prevalence of Plague in rats concurrent with the full development and activity of these insects?

The information available as to the life-history of these insects, more especially as to their season of dormancy and of activity, is, as far as I have been able to gather, of too general a nature to answer these questions. I think it would be useful if more definite information could be obtained on the subject. Perhaps some of the members who are naturalists may be able to give to the Society the benefit of their knowledge. The same interest attaches to the breeding times of the rat. The rat is, I believe, credited with three or four litters of young ones in the year. Does

this breeding time occur, as in most other animals, at a certain season of the year in the case of rats living under natural conditions? I was struck by the apparently large proportion of young rats caught during the epidemic of Plague in Hong Kong.

The next point of interest is the susceptibility of a number of the lower animals to Plague. The rat is well known for its liability to Plague, but the susceptibility of other animals not of the rodent class is not so generally recognised. During the Plague in Italy of the sixteenth and seventeenth centuries, it was a general notion, founded on observation, that dogs, cats and fowls, took the infection; but they were considered to more frequently convey the infection without themselves becoming infected. It is interesting to note that Skene remarks, in 1568, that if the domestic fowls become pestilential it is one sign of a most dangerous pest to follow.

The Chinese belief is that cattle, pigs, fowls, as well as rats, suffer from Plague. While in Hong Kong, I decided to put these views to the test of experiment. I was aware that the German Commission and Haffkine had failed to produce Plague in cats, pigs, cattle and sheep; but I have seen cats suffering from unmistakeable Plague, with Plague bacilli in their buboes; and I had before me the fact that Wilm, in 1896, had succeeded in producing Plague artificially in two pigs.

With the assistance of Dr. Hunter, the Government Bacteriologist, and Dr. Matsuda, one of the Japanese medical men lent to the Government of Hong Kong by Japan, I was enabled to ascertain that calves, hens, turkeys, geese, pigeons, sheep and pigs, were susceptible to Plague, both by inoculation and by feeding, and that pigs and poultry were susceptible to Plague in a high degree.

It appeared to me that success was more likely to follow the employment of Plague material containing the Plague bacillus and taken from a Plague case, than that of cultures of the Plague bacillus on agar or bouillon, which is a more or less artificial condition; and I attribute much of the success attained to the adoption of this method. All the material used was first carefully examined, to make sure we were dealing with Plague. Each experiment was checked bacteriologically, and by the effect produced by feeding rats on portions of the animal which had been experimented on. The bacilli were isolated, and cultures of them were made, and the effect of a few of them were tested on guinea-pigs.

There were employed in the experiment: 15 pigs, 7 calves, 1 buffalo calf, 31 hens, 6 pigeons, 6 turkeys, 6 geese, 6 ducks, 3 redbeaks, 7 monkeys, 7 guinea-pigs, and 109 rats.

It would be useless to occupy your time in entering into the details of these experiments. They are to be found in my report on the causes and continuance of Plague in Hong Kong, submitted to the Colonial Office. I shall content myself in bringing to your notice a few important points connected with them.

In the first place, a different conclusion might easily have been arrived at as regards the susceptibility of pigs to Plague, for the four pigs first experimented on—the infective material being derived from a human case—were a long time in showing signs of illness; and, if it had not been that suspicion of illness arose from the daily temperatures recorded, they would probably have been disposed of at a date anterior to their illness, and counted as failures—a fate, I suspect, of some previous experiments on pigs. Of the four pigs first experimented on, three were fed and one inoculated. The three that were fed died in the fifth week, while the one that was inoculated was killed in the fifth week. The temperatures were all much of the same type, rising on the fourteenth or fifteenth day, and continuing from that time at a higher level.

The subsequent cases were more acute. There was one case in which inoculation killed on the fourth day, while feeding with the organs of pigs which had died of Plague killed in four, eight, and thirteen days. Scarification and vaccination on the abdomen of pigs with blood and spleen pulp of other pigs which died of Plague, killed other pigs of Plague in nine and fifteen days; while the same process with the hæmorrhagic glands of a buffalo calf dead of Plague, killed in nine and nineteen days. A pig fed with the organs of a hen, which succumbed of Plague, died on the thirteenth day.

The first symptoms were usually congested eyes and unsteadiness on the legs, causing the pig to stagger or walk with difficulty, the hind legs being usually those chiefly affected. The appetite was good almost up to the last. Death, as a rule, was sudden, the symptoms of serious illness being of short duration in most of them.

The post-mortem appearances were great congestion and hæmorrhagic condition of the glands; in two cases the neck glands were the worst affected. The large intestines were congested in those that had been fed, in the others they were healthy. The bladder was always congested. Plague

bacilli were found in the blood, organs and glands, and in scrapings from the bladder. They were also in the urine of some of the pigs examined.

In the case of the calves experimented upon, the disease ran a more rapid course than that in the pig when the infection was derived from a human case, and was considerably accelerated when the infection was conveyed from calf to calf. The symptoms were as ill-defined as in the pig. There was a certain amount of dulness, the glands felt swollen and were evidently tender, and the animal lost weight. Suddenly a comotose condition would set in. The post-mortem appearances were those of congestion and infiltration, especially in the region of the neck.

Hens fed with Plague material from human Plague died on the tenth, eleventh, and fifteenth day; those inoculated died on the fifteenth day, while those inoculated or fed with material from a Plague-infected hen, or from a calf, or from a pig, or a rat, died as rapidly as the second or third day.

Turkeys, geese, and ducks, suffered from an acute or chronic form of plague: one type being fatal in a few days, the other in a month to seven or eight weeks.

An interesting experiment was the feeding of a monkey with a banana, the inside of which was smeared with the blood of a rat which had died of plague. The symptoms were in all respects similar to those induced by inoculating a monkey with the same material. Both showed a rise of temperature on the third day, with dulness, weakness, and death on the sixth day. The post-mortem appearances were general congestion of the organs of the body, congested glands without any marked enlargement, and Plague bacilli in blood and organs.

Another monkey fed in a similar fashion on another occasion remained well. The positive experiment demonstrates, however, that rat Plague is communicable to the higher animals. A similar experiment in 1896, by Wilm, in which a monkey was given a piece of sugar-cane infected by a pure culture of the Plague bacillus and died in five days, showed in the post-mortem examination a very slight swelling of the inguinal glands, great congestion of the intestines, and swelling of the mesenteric glands and of the spleen.

In another experiment a rat dead of Plague, with no visible fleas about it, but which had been opened for post-mortem examination, was placed in a cage with a monkey. The temperature of the monkey rose on the third day;

great dulness set in at the same time, which continued for three days, after which it lessened, and the monkey appeared to be getting better. There was later a relapse, and death occurred on the tenth day. There were the same post-mortem appearances as in the monkey inoculated or fed with Plague material, and there were Plague bacilli in the spleen and glands, but only a few in the blood.

The exact manner in which the monkey with the Plague-infected rat in its cage became infected it is difficult to decide. It may have been by inoculation caused by scratching, or by infection of the mouth, the fingers of the monkey becoming infected by touching the rat; or it may possibly have been due to fleas from the rat passing to the monkey, or the fleas of the monkey passing to the rat, and then again settling on the monkey.

With the object of endeavouring to settle this point, two monkeys were placed in specially-constructed cages along with rats dead of Plague, but so separated as to prevent any possibility of contact. The cages each consisted of three compartments, the middle compartment being separated from those at each end by rails which, while permitting small objects to pass between them, effectually prevented the monkey in the compartment at one end putting his hand through to reach or touch the rats in the compartment at the other end. The walls of the cages were constructed of mosquito wire netting, which prevented fleas in the cage getting outside, though they might readily pass from one compartment of the cage to the other.

In one cage a monkey was placed in one compartment, and a rat sick of Plague in the compartment at the opposite end. This rat was covered with fleas. Taken out dead three days after, there were no fleas on it. The monkey on the fourth day had a temperature of 104.6 deg. It became dull, did not eat, and was evidently sick, with its head down on its breast, and with its hand to its head; but after this illness had continued for nearly a week, it recovered. In the other case a monkey was placed in one compartment, and four dead rats in the compartment at the opposite end. The monkey on the third day had a temperature of 103.8 deg. It also became dull, and was evidently sick, but in a few days it also recovered. Dr. Hunter writes that one of these monkeys—he does not state which—died of tuberculosis fifty-two days after being experimented on. From the experiments I would advance the following propositions:—

1. That pigs, poultry and cattle are susceptible to plague,

whether derived from the infection of a human being infected with Plague, or from their own species, or from some other animal.

2. That they take infection by feeding as easily as by inoculation.

3. That Plague material from man, pigs, poultry, cattle, and monkeys will give Plague to rats, and that Plague material from rats will give plague to monkeys, by feeding, by inoculation, by contact and without contact; and if to monkeys, probably to man by the same channels.

The facility with which these animals take Plague is a very interesting point, and I am inclined to the view that Plague is not infrequently contracted by the swallowing of Plague-infected food contaminated by the infected rat, or by the uncleanliness of those preparing the food; the frequently-congested condition of the stomach and intestines of Plague patients serve to give countenance to this view.

Since these experiments were made, and more attention has been directed to the markets in Hong Kong, some of the poultry exposed for sale there have been discovered to be affected with Plague during the Plague season.

The endemicity and ill-defined character of Plague among some of the animals perhaps also explains the endemicity of the disease in those localities in which pigs and poultry share the living rooms of the inhabitants; and by their close association become, under favourable circumstances, subjected to the risk of contracting each other's diseases.

THE ELUCIDATION OF SLEEPING SICKNESS.

By Dr. LOUIS SAMBON.

(*Read: December 12th*, 1903.)

Sleeping sickness has been known to Europeans for over a century, but until quite recently we had no definite knowledge of its etiology. It was believed to be strictly confined to the negro race, and was looked upon as a peculiar form of nostalgia. At times, it caused considerable losses to the slave-traders of the West Coast, notwithstanding the careful isolation of the sick and the weeding-out of all such as presented glandular swellings about the neck. When any of the slaves showed symptoms of the disease on transport ships, they were mercilessly thrown overboard; some developed the malady long after transportation, and died of it in the plantations of the Antilles. But the disease never spread in the places to which it was imported, and therefore it did not give rise to any serious apprehension.

Within the last few years, possibly in consequence of the great commercial stir which the advent of the white man has created amongst the native tribes of Tropical Africa, sleeping sickness has begun to spread very widely, and has assumed a fearful importance in the pathology of the Dark Continent. It has extended southward throughout Angola, it has spread up the Niger and the Congo; and, proceeding along the new trade routes opened up by Europeans, it has suddenly appeared in East Africa, invading the shores and islands of the Victoria Nyanza.

Along the right bank of the Quanza, on the Upper Congo, round the northern and eastern coasts of Lake Victoria, the ravages of the dread disease have already been appalling. In many places entire villages have been depopulated.

The appearance of sleeping sickness in the very heart of Africa—the possibility of its spread along the Nile to Egypt, or by way of the coast to India; its terrible, inexorable deadliness—called for immediate and judicious action.

At the instigation of the Foreign Office, a Commission was appointed by the Royal Society to proceed to Uganda, for the purpose of investigating the cause of sleeping sickness. The Commission consisted of Drs. George C. Low,

Aldo Castellani, and Cuthbert Christy. It left London on June 10th, 1902.

In accordance with the instructions received, Dr. Low undertook to ascertain whether *Filaria perstans* had anything to do with the disease. He examined over 3000 natives, some in areas where sleeping sickness was rife, others in areas from which it was absent, and in which it had never been known; and he definitely proved that there was no causal connection between *Filaria perstans* and sleeping sickness.

Dr. Christy studied the distribution of the disease. He found that it did not prevail all over Uganda and Busoga, as reported, but that it was strictly confined to a narrow strip along the coast-line of the Victoria Nyanza, from the Kotanza river right across to Kissumu. There, it skipped a small portion, and then extended to the Gori river. The infection area did not extend for more than 10 miles from the shore. Cases beyond this limit were very rare, and the patients had always a history of having come from the lake shore. The affected area included all the islands, many of which were taxed severely. Dr. Christy made a trip to Buvuma Island, and there mapped all cases in the places in which they occurred. The dots on the map showed that the distribution of the disease on the island corresponded to the general distribution of sleeping sickness. It was confined to the coast-line overgrown with bush, chiefly bananas. Dr. Christy also noticed that there was no connection between *Filaria perstans* and sleeping sickness. He observed that the disease extended about 150 miles beyond the extreme limit of *Filaria perstans*.

Meanwhile, Dr. Castellani isolated a *Streptococcus* from the blood and cerebro-spinal fluid of patients suffering from sleeping sickness. At first he conjectured that this bacterium might be the cause of the disease, but later he noticed that it was found rarely during life, and then only in the last stages.

On November 12th, 1902, while examining some cerebro-spinal fluid, obtained from a case of sleeping sickness by means of the lumbar puncture performed during life, Castellani discovered a *Trypanosoma*. At the moment, he did not attach much importance to this finding. He considered the presence of the flagellate a mere coincidence, and thought that it had probably arisen from admixture of blood during puncture. Subsequently, however, he observed that trypanosomes, though always few in number, were of frequent occurrence in the cerebro-spinal fluid of patients suffering

from sleeping sickness; and he communicated his discovery to Drs. Moffat and Baker, and afterwards to Lieut.-Colonel Bruce, as soon as the latter arrived in Uganda.

Dr. Castellani's discovery was followed up very rapidly by a number of observers; and thus, in a twelvemonth, the etiology and epidemiology of sleeping sickness have been almost entirely worked out, and the deadliest scourge of Africa is under the heel of the white man's foot.

The history of this discovery is a most brilliant proof of the exactness of modern scientific medicine; and the famous boast, "*Veni, vidi, vici!*" could not find a more appropriate application.

Although we are indebted to Dr. Castellani for the discovery of the *vera causa* of sleeping sickness, it is but fair to state that the complete elucidation of the disease has been the work of numerous observers; and we should not forget the researches of the old English and French surgeons who gave us the first accounts of the malady, any more than the latest and most important investigations by Bruce, Brumpt, and Manson. Our modern triumphs have deep roots, not only in the discoveries, but also in the mistakes of the past. The old dictum, "*Errando discitur*," applies to the advancement of science just as truly as it applies to the experience of life.

So far as I know, the first to describe sleeping sickness was Dr. T. Winterbottom, in *An Account of the Native Africans in the Neighbourhood of Sierra Leone*, which he published in London exactly a hundred years ago. Then followed Papers by Clarke, Dangaix, Nicolas, Gaigneron, Karl, Griffon du Bellay, and Santelli, who described fairly well the symptoms and the coarse anatomical features of the disease.

In 1869, Dr. P. A. Guérin published an excellent thesis on sleeping sickness. During a period of about twelve years in Martinique, Guérin had the opportunity of studying 148 cases of the disease in Negroes imported from the Congo. In some cases the period of incubation lasted five years, or more. The disease did not spread to the black Creoles of the Antilles; neither did it spread to other Congolese who had been in the colony some forty years. In his Report to the Royal Society, Dr. Christy questions the correctness of Guérin's observations; but his criticism is futile. Besides, as he owns himself, he read only a summary of Guérin's Paper in the *Archives de Médicine*. The so-called incubation period of sleeping sickness, like that of hydrophobia, may certainly extend over a period of two,

three, or more years. A case occurred in England in a Congo boy, who had resided in this country for three years without showing any particular signs of unhealthiness.

In 1876 and 1877, Dr. A. Corre published some very interesting Papers on sleeping sickness. Corre had made a special study of the disease in the severely-affected districts of Joal and Portudal in Senegambia. He laid special stress on the paludal nature of the sleeping sickness centres, and remarked that the disease may attach itself to a particular house or to a particular group of houses. He referred that a number of patients believed they had contracted the disease during the rainy season. In his first Paper, Corre conjectured that the disease might be a kind of food-poisoning, analogous to ergotism and lathyrism; but later he put it down to scrofula, on account of the frequent occurrence of glandular swellings. Corre gave an admirable description of the symptoms of the disease. He noticed that, in most cases, a regular evening rise is the characteristic temperature; he also noticed an opacity of the cornea in some cases.

In 1891, while examining the blood of a case of sleeping sickness under the care of Sir Stephen Mackenzie in the London Hospital, Sir Patrick Manson discovered the larvæ of a new filaria, which he named *Filaria perstans*, because of the constant presence of its young in the peripheral circulation. Subsequently, he found the larvæ of *Filaria perstans* in films of blood obtained from cases of sleeping sickness on the Congo; and, in 1898, he again found them in two cases of sleeping sickness which Dr. Grattan Guinness brought over to England for the purpose of study, and which were admitted into Dr. Abercrombie's ward at Charing Cross Hospital. Struck by the constant presence of these blood-worms in cases of sleeping sickness, and by the singular corespondence which seemed to exist between the geographical distribution of sleeping sickness and that of *Filaria perstans*, and paralleling the long incubation period of sleeping sickness with the fact that *Filaria perstans* can remain alive within the body of its host years after the infection area has been quitted, Manson suggested that this Filaria might possibly be the cause of sleeping sickness. When it became evident that *Filaria perstans* was present not only in cases of sleeping sickness, but also in a large proportion of the normal inhabitants of the Congo and of other parts of West Africa, Manson still held to his hypothesis, because it was supported by the pathology of *Filaria bancrofti*. In fact, *Filaria bancrofti*

does not always cause chyluria; yet there can be no doubt that it is, within the Tropics, and in many sub-tropical countries, a frequent cause of chyluria.

The discovery of the presence of *Filaria perstans* in British Guiana was decidedly against the theory of a connection between this nematode and sleeping sickness; but the diseases of the natives of British Guiana are very little known, and Dr. Ozzard wrote that he believed sleeping sickness to be amongst their ailments; indeed, he stated that a Buck woman was brought to him for treatment, because, as her friends said, she was always asleep.

Low's recent researches in British Guiana, and the numerous and accurate observations he made in various parts of Uganda, have definitely proved that there is no causal association between *Filaria perstans* and sleeping sickness.

Drs. Hodges and Wiggins had already shown, before the Commission arrived in Uganda, that, though sleeping sickness was very prevalent in the Kavirondo Islands, *Filaria perstans*, on the other hand, was very rare. Besides, the distribution of *Filaria perstans* is very wide throughout intertropical Africa, wherever the œcological conditions are favourable. But all this information is very recent.

The suggestion that *Filaria perstans* might be the cause of sleeping sickness has proved erroneous; but the suggestion was very reasonable; and, indeed, it led to the discovery of the true cause of the disease, just as an analogous mistake led to the discovery of America.

Other nematodes have been incriminated as causative agents in sleeping sickness, but on very poor grounds. Forbes ascribed the disease to *Strongyloides intestinalis;* Fergusson to *Uncinaria duodenalis.* Both worms have a very wide distribution. *Uncinaria* is almost cosmopolitan.

It would be idle to discuss the various theories put forward by the older authors to account for this strange and formidable disease. Some believed it to be a kind of nostalgia, intensified by the ill-treatment suffered at the hands of slave-traders and planters. Others ascribed it to malaria, to sunstroke, to amenorrhea, to inanition, to the immoderate drinking of palm wine, to the eating of raw manioc, or to the smoking of Indian hemp.

The food-intoxication theory, suggested first by Corre, then again by Pereira do Nascimento and Calmette, was revived last year by Ziemann, who ascribed sleeping sickness to the eating of raw or unsuitably-prepared manioc. Ziemann says that he was led to this theory by a previous

study of pellagra and beri-beri, which, he affirms, are known to be diseases of intoxication. In controverting Ziemann's theory, I must begin by stating that his premise is wrong. So far, no one has actually proved that pellagra is caused by some specific toxic substance contained normally in maize; neither has anyone proved that beri-beri is due to an analogous poison in rice. Both maize and rice are perfectly healthy foods under ordinary conditions; and, whatever their part may be in the natural history of pellagra and beri-beri, certainly they do not stand in the direct relation of cause and effect. Like many other food-plants now extensively cultivated in West Africa, the bitter cassava (*Manihot utilissima*) and the sweet cassava (*Manihot aipi*) were imported from South America. The areas of their cultivation and consumption in Africa do not in any way coincide with the geographical and topographical distribution of sleeping sickness. Another reason against the manioc theory is the occurrence of sleeping sickness among Negroes far and long removed from the endemic centres of the disease, as in the West Indies and in Europe.

Various kinds of bacteria have been described in connection with sleeping sickness, and claimed to be the specific agents of the disease. In 1897, Cagigal and Lepierre found a bacillus in the blood of a case of sleeping sickness imported from Angola, and claimed that it was the cause of the disease. They stated that, by inoculating rabbits with cultures of this bacterium, they produced a disease resembling sleeping sickness, and yielding the characteristic organism. Brault and Lapin, who procured a culture of the bacillus, were unable to confirm these observations.

In 1899, Marchoux suggested that *Fränkel's diplococcus* might be the cause of sleeping sickness. He performed the autopsy of one case of the disease at Saint Louis (Senegal) and found a *Diplococcus* on the pericardium, but was unable to detect its presence within the cerebro-spinal system. Pneumonia was very prevalent at the time.

In 1901, Broden examined several cases of sleeping sickness at Leopoldville (Congo), and found in the blood and in the cerebro-spinal fluid (*post-mortem*) a bacillus which grew abundantly on potatoes. This bacillus, possibly *Bacillus solanacearum*, was not agglutinated by the blood of patients suffering from sleeping sickness.

The same year, the Portuguese Government sent a Commission to Angola to investigate the etiology of sleeping sickness. Bettencourt and his colleagues isolated a *Streptococcus* from the cerebro-spinal fluid obtained by means of

the lumbar puncture performed during life, or *post-mortem*. The lumbar puncture was performed in nine cases. In six of these it gave positive results, the bacteria being easily isolated; in the other three, both the examination of the direct preparations and of the cultures gave negative results. The *streptococcus* was also found in the blood and in the lymph glands.

Castellani also found a *streptococcus* in the blood and cerebro-spinal fluid of patients suffering from sleeping sickness, but only in the last stages of the disease. Indeed, he grew it once only from the blood, although he examined bacteriologically the blood of thirty-seven patients, and in each case repeated the investigation several times, and with different methods. He examined the cerebro-spinal fluid obtained by lumbar puncture in twenty-eight patients, but only five cases gave a positive result, and four of these were examined a few hours before death. Out of six bacteriological examinations of urine, he grew the microbe once. Bacteriological examinations of enlarged lymphatic glands removed during life were negative, and the examination of the spleen juice obtained by puncture during life was likewise negative.

Probably, Castellani's *streptococcus* is identical with the microbe described by the Portuguese Commissioners. The latter first stated that their *Diplo-streptococcus* grew very poorly on the ordinary culture media, and that they had never succeeded in obtaining cultures on gelatine. After Castellani's publication, however, they modified their statements, and affirmed that their *streptococcus* grows very well on gelatine.

Castellani believes that the *Streptococcus* he has found in cases of sleeping sickness is merely a variety of *Streptococcus pyogenes*. He is inclined to think that this organism may play a part in the etiology of sleeping sickness, similar to that of the *streptococci*, associated with the complications of scarlet fever and rheumatic polyarthritis.

The bacteria so far described in connection with sleeping sickness have probably no casual connection with the disease; they are seldom found in the early stages of the disease; they are often absent in the last. The two cases of sleeping sickness which were brought to London in 1898 were very carefully examined with regard to bacteria. Dr. Bullock attempted to make cultures from the blood and from some enlarged cervical glands which were removed during life, but none of the cultures had grown at the end of three weeks; he therefore came to the conclusion that no ordi-

nary micro-organisms were contained in the blood or glands. At *post-mortem* the cerebro-spinal fluid was examined for micro-organisms by cultures, etc., and various organisms, including *diplococci*, *streptococci*, and *bacilli* were found; but, of course, no importance could be attached to these observations.

The question of the *rôle* played by *streptococci* in scarlet fever and other infections is a very difficult one, and recent researches by Aronson and others show that we are further than ever from its solution.

A very important step in our knowledge of sleeping sickness was the study of its minute anatomical features. The first detailed microscopical examination was that which Regis and Gaid published in 1898. These authors correctly attributed the symptoms of sleeping sickness to a diffuse meningo-encephalitis; but their observations referred to a single case in the region of Timbuctoo.

In 1900, Dr. Frederick Mott published the changes he had found in the central nervous system of two cases of the disease, and remarked that sleeping sickness is due "to a poison of micro-parasitic or other source, which affects especially the lymphatic system, and in particular that portion of it pertaining to the central nervous system." Mott's excellent observations were confirmed by the Portuguese Commissioners in 1901, and by Warrington in 1902.

The chief characteristics after death from sleeping sickness are: general emaciation, enlargement of the lymphatic glands, slight opacity and thickening of the pia-arachnoid, and serous effusion into the meshes of the pia-mater or into the ventricles. The microscope reveals an intense chronic meningo-encephalo-myelitis.

The emaciation is very marked in certain cases, especially on account of starvation. The enlargement of the lymph glands is constant, and may be noticed in the cervical, axillary, mesenteric and inguinal groups. In the cerebro-spinal system the macroscopical changes are seldom marked. In most cases there is only a slight opacity of the pia-arachnoid over the convexities, and some serous exudation in the sub-archnoidal space. In some rapid cases the exudation may be considerable in amount. As a rule, it is slightly turbid, but never purulent unless the disease be complicated by *streptococcic* invasion. The vessels of the brain do not show any appreciable abnormality, but there may be a marked congestion of the arteries and veins of the dura mater. The cerebro-spinal fluid may be slightly turbid, but seldom in excess. The ventricles are rarely dilated.

The brain-substance is of normal consistence, and, as a rule, the convolutions are neither flattened nor wasted. *Puncta cruenta* are rarely marked in the cut surface of the cerebrum. The medulla shows to the naked eye marked congestion of the vesssels.

The microscope shows the pia-arachnoid infiltrated with mononuclear leucocytes; the inflammation is seen throughout the whole central nervous system, but especially in the medulla and at the base of the brain. It can be traced along the blood-vessels and septa into the substance of the nervous system. The perivascular lymphatics around both large and small vessels are crowded with these lymphocytes. The cells of the cerebral cortex show a normal outline; but scattered through the substance, especially in the pericellular spaces, are the small round nucleated cells.

The enlargement of the lymph glands throughout the body, and the enormous accumulation of mononuclear leucocytes within the perivascular lymphatics of the cerebro-spinal system, indicate that the specific agent of sleeping sickness is essentially a parasite of the lymphatics; but that it is capable of damaging the nervous elements, either by mechanical action, or possibly by elaborating a special toxin.

Castellani's discovery of a trypanosome in the cerebro-spinal fluid of patients suffering from sleeping sickness marked a new era in the study of this fearful disease, and suggested a very definite line of research with regard to its etiology, epidemiology, and prophylaxis.

For some years trypanosomes had been the object of very active researches, and a large amount of knowledge had been accumulated. Castellani's discovery brought all this knowledge to bear on sleeping sickness; and, thus in less than a year, the elucidation of sleeping sickness becomes the boast of modern scientific medicine.

The genus *Trypanosoma* was established by Grubey in 1843; but the wimble-like *hæmatozoa*, included in this division, were first described in 1841 by Valentin, who discovered them in the blood of a trout (*Salmo fario*), and the following year by Gugge, who found them in frogs. Since then numerous species have been described from fish, batrachians, and birds.

The first to find these parasites in mammals was Dr. Timothy Lewis, who, in a sadly short career, has so largely contributed to the advancement of Tropical medicine. Lewis found them in 1879 at Calcutta, in the blood of rats (*Mus decumanus* and *Mus rufescens*). The rat-trypano-

some, very appropriately called *Trypanosoma lewisi*, has a world-wide distribution corresponding with the cosmopolitanism of its mammalian host. But its prevalence varies greatly in different localities. The researches of Rabinowitsch and Kempner, and those of Laveran and Mesnil, have greatly advanced our knowledge of this flagellate. Similar parasites have been found in hamsters, guinea-pigs, rabbits, and other rodents.

In 1880, in the Punjab, Griffith Evans discovered in the blood of horses a trypanosome of a far greater pathological importance. The horses examined by Evans were suffering from Surra, a disease known to the natives of India from time immemorial, and ascribed by them to the bite of certain *Tabanidæ*. The Surra parasite (*Trypanosoma evansi*) is not limited to horses and mules, but attacks also camels, elephants, buffaloes and dogs. Experimentally it has been transferred to monkeys, rabbits, rats, mice and guinea-pigs. "Surra" has a very wide distribution in Southern Asia and in Malaya. It has been described by the Germans in East Africa, and, quite recently, by the Americans in the Philippine Islands. Evans was favourable to the popular opinion which ascribed the transmission of the disease to certain blood-sucking flies; but subsequent authors, and especially Lingard, considered food and drinking-water to be the true vehicles of the infection.

The discovery of a trypanosome in Surra, the fly-disease of India, was followed by the discovery of a trypanosome in Nagana, the fly-disease of Africa.

The fly-disease of Africa has been known as long as the fly-disease of India. James Bruce, in his famous *Travels to Discover the Source of the Nile*, mentions a blood-sucking fly, probably a tsetse fly, under the name of "Tsaltsalya," or "Zimb," and describes its terrible ravages amongst horses and cattle, during the rainy season, on the Upper Atbara river, on the confines of the Sudan and Abyssinia. In South Africa "Nagana" has long been known to Europeans as the "Fly-disease."

Dr. Livingstone, in his *Missionary Travels and Researches in South Africa*, gives much information regarding tsetse flies and the disease they inoculate. After describing the symptoms of Nagana, he very correctly says: "These symptoms seem to indicate what is probably the case—a poison in the blood, the germ of which enters when the proboscis is inserted to draw blood. The poison-germ, contained in a bulb at the root of the proboscis, seems capable, although very minute in quantity, of reproducing itself."

In 1879, Dr. J. J. Drysdale, after referring to Manson's discovery of the transference of *Filaria bancrofti* by mosquitoes, says :—"It is possible that we have here an explanation of the destructive power of the tsetse fly, for it may be the intermediate host of some similar blood parasite; or it may be the carrier of some infective poison. It is highly improbable that any mere poison or venom should exist so powerful as to cause the death of a large animal in such small dose."

A Swiss entomologist named Schoch, in 1883, and A. Laboulbène, in 1888, again expressed the opinion that the tsetse is not poisonous in itself, but that it acts as a carrier of pathogenic micro-organisms.

In the short time at my disposal it would be impossible for me to mention the numerous authors who have contributed to our knowledge of the African fly-disease. These authors described very accurately the symptoms of Nagana —its coarse anatomical lesions, its peculiar distribution, the immunity of local wild animals, the structure, distribution, and life-habits of its carrier, the tsetse fly.

At last, in 1895, Surgeon-Major—now Lieut.-Colonel—David Bruce went to Zululand to investigate Nagana, and found that the specific parasite of the African horse-disease was a trypanosome similar to the one found by Evans in Surra fifteen years previously. Drs. Kauthack, Durham, Blanford, Plimmer and Bradford made a more complete study of the parasite, and the latter two named it *Trypanosoma brucei*. Then followed numerous investigations by Laveran and Mesnil, by Koch, by Rabinowitsch and Kempner.

Lieut.-Colonel Bruce made several experiments to prove that the disease was transmitted from infected to normal animals by means of the bite of tsetse flies. This point, however, had already been investigated some time ago. In fact, in 1857 Livingstone wrote: "The curious feature in the case—that dogs perish, though fed on milk, whereas the calves escape so long as they continue sucking—made us imagine that the mischief might be produced by some plant in the locality, and not by the tsetse. But Major Vardon, of the Madras Army, settled that point by riding a horse up to a small hill infested by the insect without allowing him time to graze; and though he only remained long enough to take a view of the country, and catch some specimens of tsetse on the animal, in ten days afterwards the horse was dead."

In Southern Europe, and right round the Mediterranean

basin, we also find a trypanosome infection peculiar to horses, and known since the beginning of the nineteenth century under the names of Dourine and *Maladie du coit.* The parasite *Trypanosoma equiperdum* was first discovered by Chauvrat in 1892.

Another trypanosome disease of horses is the *Mal de caderas*, which ranges over a wide area in South America. The parasite was discovered in 1901 by Dr. Elmassian, an old student of the Institut Pasteur. Voges gave it the name of *Trypanosoma equinum.*

To complete the list of trypanosomes so far described in the domesticated mammals, I must mention the large trypanosome found by Theiler in 1903, in the so-called "gall-sickness" of South African cattle, and named by Colonel Bruce *Trypanosoma theileri.*

A very important question is to determine whether these variously-named and variously-distributed diseases are distinct morbid entities or not.

The "gall-sickness" of South Africa appears to be a specific disease affecting only cattle. Its parasite, *Trypanosoma theileri*, attains greater dimensions (30—65 μ) than the other trypanosomes.

We should not forget, however, that the same species of *trypanosoma* may present very different dimensions in different host-species. Thus, *Trypanosoma brucei*, which measures about 26 μ in the rat, attains a length of from 28 to 33 μ in the horse.

Mal de caderas seems likewise to be a separate disease. Its parasite *Trypanosoma equinum* is easily distinguished from the other trypanosomes by the fact that its micronucleus is almost invisible on account of its very small size and does not acquire a deep colouration in stained specimens, but stains faintly, like the flagellum.

As to the trypanosomes of Surra, Nagana, and Dourine it is not possible to distinguish them morphologically Dourine seems to differ somewhat symptomatically from Surra and Nagana. Dogs may recover from its attacks, and the animals which have acquired immunity against Dourine are not immune to Surra or Nagana.

Surra and Nagana are strikingly alike. They affect the same animals, they give rise to the same symptoms, they cause the same anatomical lesions. Their identity, suggested by Koch, Schilling and Rogers, seems, therefore, quite possible.

In distinguishing between trypanosomes, much importance has been attached to experiments of inoculation into

other animals; but we should not forget the frequent fallaciousness of such experiments. Different investigators have often obtained opposite results. Different species of oxen, horses, or dogs may exhibit a very different degree of susceptibility. Then again the passage of the parasite through certain animals may greatly alter its virulence, and even modify its aptitude for the original host. This all-important fact, which has been thoroughly ascertained in the laboratory, should not be lost sight of in epidemiological investigations.

It is a general law in parasitism that parasites become more and more adapted to the host-species they repeatedly inhabit. In time, they become so specialised—so greatly modified and changed—that they are absolutely dependent upon such hosts, and are unable to thrive in any others. Sometimes the limitation to certain hosts is not associated with any apparent structural change; and naturalists have proposed to call "biologic species" those species which cannot be distinguished by morphological characters.

An example, which shows how such limitations may arise, is that of the stem eel-worm (*Tylenchus devastatrix*). This nematode lives and reproduces in various cultivated plants, such as rye, oats, stored onions, hyacinths, buckwheat, potatoes, and clover; and in wild plants such as *Poa annua*, *Anthoxanthum odoratum*, *Dipsacus silvestris*, and *Polygonum persicaria*, but not to the same extent in all. However, eel-worms of which the progenitors have developed for many years exclusively in rye and buckwheat are not easily transferred to another kind of plant; or, at any rate, they do not multiply vigorously there.

Various blood-sucking insects have been incriminated as carriers of the different trypanosome infections, but the information so far at hand is very scanty and most unsatisfactory. Rat-fleas are believed to be the carriers of *Trypanosoma lewisi*, because living trypanosomes have been found in the mid-gut of these insects. McNeal and Novy saw living specimens of *Trypanosoma lewisi* in the stomach of rat-lice. Dourine is generally believed to be acquired by direct contact with the secretions of the œdematous genital mucosae of infected animals; but Rabinowitsch and Kempner found the trypanosomes of Dourine in the stomachs of fleas. They think, therefore, that Dourine may be also due to the agency of blood-sucking insects. For the Surra trypanosome certain horse-flies (*Tabanidae*) have been incriminated by Rogers as its carriers in India. Curry found the Surra trypanosome of

the Philippines in the mid-gut and proboscis of *Stomoxys calcitrans*. In 1898, Captain A. C. Haslam, at Machakos, found the living trypanosomes of Nagana in the stomach of specimens of two species of *Stomoxys*, caught sucking the blood of mules suffering from the disease. Lignières found the trypanosomes of *Mal de caderas* in the stomach of *Stomoxys calcitrans*, and Sivori and Lecler stated that they allowed sound horses to be bitten by these flies, and obtained positive results. Lignières, however, was unable to obtain infection of horses with *Mal de caderas*, by means of either *Tabanidae* or *Stomoxys calcitrans*. Lastly, Theiler found living trypanosomes in the stomach of specimens of *Hippobosca rufipes* and *Hippobosca maculata*, fed on cattle suffering from "gall sickness." He decided, therefore, to try the infection of cattle by the medium of the fly's bite. For this purpose some flies were fed on the groins of an infected calf, and then placed on a healthy animal. To give the experiment every chance of success, the place where the flies were put to feed was first shaved, as was also the spot on the normal animal where they were placed for infection. Feeding by turns on a sick and on a clean animal was thus repeated several times. Out of four experiments made in this way, two were successful.

Turning now to the discovery of trypanosomes in man, we find that the first to describe trypanosomes in man was Dr. Nepveu.

Dr. Nepveu found these flagellates in Algeria, in 1890, while studying malaria parasites. In a paper published on December 24th, 1898, in the *Comptes Rendus des Séances de la Société de Biologie*, he says: "This trypanosome presents all the characters of the genus; general shape, a homogeneous colourless membrane, one border of which is thinner, hyalin, and presents characteristic undulatory movements. This membrane bears a nucleus, and a slender flagellum which is placed anteriorly, and the undulations of which follow in rapid succession." Nepveu describes segmentation forms with two flagella at one of their extremities, and correctly considers them to represent a developmental stage of the trypanosome. "In conclusion," he says, "trypanosomes must be classed amongst the parasites of human blood."

On May the 10th, 1901, at Bathurst, Gambia, Mr. R. M. Forde, while examining the blood of a patient suffering from an anomalous form of fever, discovered some small worm-like, actively-moving bodies, the nature of which he was unable to ascertain. The patient, master of a

Government steamer on the Gambia, went home on sick leave, and on August 12th entered the Royal Southern Hospital, Liverpool, where he suffered from "three short periods of pyrexia," and other symptoms. Here Dr. J. Everett Dutton examined his blood for malarial parasites on two occasions, with negative results. Having considerably improved, the patient returned to Bathurst in the early part of December, 1901. At about the same time, Dr. Dutton also arrived at Bathurst, and Mr. Forde asked him to examine the blood of the patient once more, because he was sure it contained some extremely active worm-like bodies, which might prove of great pathological interest. On doing so, Dr. Dutton, who had previously found nothing in the patient's blood, now succeeded in detecting the parasite, recognised it to be a trypanosome, and named it *Trypanosoma gambiense.*

To Mr. Forde we are indebted not only for the discovery of trypanosomes in Gambia, but also for the first description of the symptoms peculiar to the early stage of *Trypanosoma* infection.

The description of the parasite and the clinical symptoms of Forde's patient were published first by Dr. Dutton in the Thompson-Yates Laboratory Reports, and later by Forde himself in the *Journal of Tropical Medicine.*

On October 3rd, 1902, Sir Patrick Manson examined a lady, the wife of a missionary who had just returned from Monsambe on the Upper Congo. Struck by the fact that the symptoms presented by this patient (daily recurring fever, enlarged spleen, patchy œdema, erythema multiforme) closely resembled those he had noticed two months previously in Forde's case, he made a provisional diagnosis of *Trypanosoma* fever, Manson's diagnosis was subsequently confirmed by Dr. Daniels who, after a long search, found trypanosomes in the blood of this patient.

Other cases were soon afterwards described by Drs. Broden and Brumpt on the Congo. Meanwhile, a commission consisting of Drs. J. E. Dutton and J. H. Todd was despatched to the Gambia by the Liverpool School of Tropical Medicine, to ascertain the extension and prevalence of the disease in the colony. Dutton and Todd examined over a thousand persons, and found the parasite in six natives and one white man (quadroon). The history and clinical examination of the native cases revealed nothing important. In the younger cases the usual enlargement of the spleen was met with. Enlargement of the lymphatic glands was present in some, but not in

others; parasites were very scanty. The disease, as it occurred in natives, was a peculiarly mild one. This absence of marked clinical features in natives corroborates Nepveu's observations, because the latter failed to recognise in his patients any symptoms characteristic of *Trypanosoma* infection. I mention this because, in an article contributed to the *Lancet* on February 21st, 1903, Professors Boyce, Ross and Sherrington endeavoured to disprove Nepveu's observations, and pointed to the latter's statement of the non-pathogenetic nature of the parasites as a strong argument against the veracity of his observations.

Drs. Dutton and Todd examined various animals in Gambia, to ascertain whether any were infected by trypanosomes, but this parasite was found only in horses. Out of thirty-six horses examined ten were infected. The symptoms noticed were: loss of vigour, periodical rises of temperature, marked emaciation, and, at times, a slight watery discharge from the eyes. The diseased animal wore an apathetic, chronically-tired expression which was most characteristic. In none of these horses did they observe the marked œdemas and the changes in the coat described as characteristic of Nagana. There was a general enlargement of all the lymphatic glands.

No morphological distinction could be made between *Trypanosoma gambiense* and the horse trypanosome. Therefore, it was impossible to decide whether the parasites found originally in the horse and man were the same or different. Several experiments of inoculation into lower animals were made with both parasites. These experiments seemed to indicate a marked distinction between *Trypansoma gambiense* and the horse parasite.

As soon as Castellani discovered a trypanosome in the blood and cerebro-spinal fluid of patients suffering from sleeping sickness, the idea of a fly as the carrier of the infection came very naturally to all those who had any knowledge of *Trypanosoma* diseases.

The first to publish any definite opinion on the matter were Dr. Brumpt and myself. It would be idle to discuss priority in the matter. I expressed my views in a lecture on sleeping sickness, delivered at the Livingstone College, and subsequently published in the *Journal of Tropical Medicine* of July 1st, 1903. Dr. Brumpt's observations were published on July 2nd. However, the idea came to both simultaneously and independently the day we learnt that a trypanosome was possibly the cause of sleeping sickness.

Dr. Brumpt incriminated *Glossina morsitans*; I suggested a West African species, and more especially the widely-distributed *Glossina palpalis*; but I attach no great importance to this, because Brumpt was in the wilds of the Congo, and therefore unable to identify his specimens. Besides, various species of the genus *Glossina* may be capable of transmitting sleeping sickness just as various species of the genus *Anopheles* are able to disseminate sub-tertian fever. Our respective communications did not merely suggest the possibility of a *Glossina* as the carrier of sleeping sickness, because a trypanosome had been described in connection with sleeping sickness, and because Nagana was known to be disseminated by a *Glossina*; but, inversely, they were intended to support Castellani's discovery by a number of facts gleaned from a careful study of the distribution and life-history of tsetse flies, as well as of the distribution and epidemiology of sleeping sickness.

At first, Castellani's discovery met with little favour in this country. At the last meeting of the British Medical Association, in Swansea, when the subject of trypanosomiasis was thoroughly discussed, I was the only one to uphold a causal relationship between Castellani's trypanosome and sleeping sickness. My opinion was based on the many analogies between sleeping sickness and the various *Trypanosoma* diseases of cattle in their symptoms, course, and anatomical features; and more especially on the striking connections which exist between the peculiar distribution of sleeping sickness and that of tsetse flies, between the epidemiology of the disease and the bionomics of the *Glossinæ*.

I stated quite clearly that I could see no morphological difference between Castellani's trypanosome and *Trypanosoma gambiense*. I pointed out that the so-called anterior extremity of trypanosomes is really their posterior extremity, and that the true anterior extremity is the one which encloses the micro-nucleus, and from which arises the flagellum. The most cursory examination of the various flagellates, and more especially of the free-living forms, suffices to prove that the tapering end of the trypanosome, continued by the free extremity of the flagellum, is the posterior extremity. Authors have called it "anterior," because the parasite moves more frequently and more rapidly with this extremity foremost; but the mode of progression is not a sound argument, otherwise we should have to term "posterior extremity" the well-defined head

of cephalopods, because these animals are capable of a very rapid backward motion, and in this way they usually escape the animals that prey upon them.

I also expressed the opinion that the *Glossinæ* do not merely transfer the trypanosomes in a passive way, as suggested by Lieut.-Colonel Bruce, but that, somehow, they play the part of alternative hosts in their propagation.

Considering the very remarkable mode of development which obtains in tsetse flies, I further suggested that both Nagana and sleeping sickness might be transmitted, not directly by the fly that sucks the blood of infected animals, but by its progeny, as is known to be the case in the transmission of Red-water fever by the cattle tick, *Rhipicephalus annulatus*, and in the transmission of the malignant jaundice of dogs by another tick: *Haemaphysalis leachi*.

In August, the Royal Society published a Progress Report, by Lieut.-Colonel David Bruce and Dr. David Nabarro. These two gentlemen fully confirmed Dr. Castellani's discovery. They stated that whilst Dr. Castellani had found the trypanosome in about 70 per cent. of cases, they found it almost in every case of the disease, even in the early stages; and not only in Uganda, but also in other districts affected by the sickness.

Castellani had already observed that trypanosomes were not present in the cerebro-spinal fluid of natives suffering from other diseases, but free from sleeping sickness. Bruce and Nabarro examined fifteen more cases, and proved the statement correct. Dr. Wiggins also examined seven natives free from sleeping sickness in Kissumu, and likewise failed to find trypanosomes.

Castellani had found the trypanosome also in the blood of one of his cases of sleeping sickness. Bruce and Nabarro asserted that the presence of trypanosomes in the blood could be demonstrated almost in every case of sleeping sickness.

As cases of "*Trypanosoma* fever" had been described by Drs. Baker and Moffat in Uganda, Bruce and Nabarro considered the question of a possible connection between the two conditions. However, they did not venture any definite opinion, but merely described certain morphological differences, which, they thought, seemed to separate Castellani's trypanosome from *Trypanosoma gambiense*. They stated that the flagellate found in sleeping sickness was shorter, had chromatic dots more frequently, and the

micro-nucleus was situated nearer the extremity than in *Trypanosoma gambiense.*

Numerous experiments were carried out in monkeys, dogs, rats, and other animals, both with trypanosomes from sleeping sickness cases and from *Trypanosoma* fever. Both sets of experiments gave positive results, but did not lead to any definite conclusion.

From the analogy suggested by Nagana, Bruce and Nabarro suspected that a tsetse fly might be the carrier of the disease. Two "varieties" of *glossina* were obtained from the Botanical Gardens at Entebbe, and experiments at once commenced. One monkey, after having been bitten on successive days by 215 tsetse flies, showed trypanosomes in its blood 14 days after the first day of experiment.

In November appeared a "Further Report" by Lieut.-Colonel Bruce, Dr. Nabarro, and Captain Greig. In this report the Commissioners stated that they were inclined to consider *Trypanosoma* fever as the first stage of sleeping sickness. They described two cases of *Trypanosoma* fever which, after a short period of improvement, developed symptoms suggestive of sleeping sickness. At the same time, the parasites, which had been confined to the blood, appeared in the cerebro-spinal fluid.

In this second report the Commissioners retracted their previous statement concerning the morphological differences between *Trypanosoma castellanii* and *Trypanosoma gambiense.* They stated that the differences mentioned in the Progress Report have no specific importance, but only show that the parasites find the cerebro-spinal fluid not so favourable for their growth as the blood. Trypanosomes from the cerebro-spinal fluid injected into the blood of monkeys became quite as long as the trypanosomes found in the blood of man.

Numerous experiments were described in monkeys, dogs, guinea-pigs, donkeys, oxen, sheep, and goats. The experiments were carried out with trypanosomes taken both from cases of sleeping sickness and *Trypanosoma* fever. The inoculations were performed into the brain, into the vertebral canal, or sub-cutaneously. The monkeys showed the greatest susceptibility, and exhibited symptoms resembling those of sleeping sickness, no matter whether inoculated with the cerebro-spinal fluid of sleeping sickness cases or the blood of *Trypanosoma* fever cases. Rats and dogs were only partially susceptible; oxen, donkeys, goats, sheep and guinea-pigs were absolutely refractory. These experiments tended to prove the identity of the

trypanosomes found in sleeping sickness and *Trypanosoma* fever.

Other experiments were made to determine whether the Uganda tsetse flies are able to convey sleeping sickness.

Five monkeys were submitted to the bites of tsetse flies. The flies were collected in the neighbourhood of Entebbe; they were fed on patients suffering from sleeping sickness, and after eight, twenty-four, or forty-eight hours they were made to bite the monkeys. Hundreds of flies were used in each experiment, and the monkeys were bitten every day by ten, thirty, sixty or a hundred and forty flies until a positive result was obtained.

A second set of experiments with tsetse flies and monkeys was also carried out, to ascertain whether flies freshly caught in the vicinity of Entebbe were actually carrying the trypanosomes of sleeping sickness.

Three monkeys were submitted to the bite of large numbers of flies caught in the vicinity of the hut-tax labourer's camp in Entebbe. The flies were fed only on the monkeys soon after being caught.

Both sets of experiments gave positive results, with a very striking difference, however; because, while trypanosomes appeared in the blood of the monkeys eight weeks after the first bite, in those animals which were bitten by flies previously fed on cases of sleeping sickness, they appeared after only two weeks in those which were bitten by flies not artificially infected.

From these experiments the Commissioners concluded that they had proved:—

(1) That *Glossina palpalis* is capable of conveying trypanosomes from sleeping-sickness cases to healthy monkeys, up to at least forty-eight hours after feeding.

(2) That the flies collected in the vicinity of Entebbe were actually carrying the trypanosomes of sleeping sickness.

I dispute the conclusions arrived at by Colonel Bruce and his collaborators. Their experiments did not prove that the flies conveyed the trypanosomes of sleeping sickness from the patients on which they were fed to the healthy monkeys. On the contrary, the presumed artificial infection only served to deprive the flies of the majority of the trypanosomes they were harbouring. From the positive results obtained in the second set of experiments, we learn that the flies were already infected with trypanosomes when caught. When the flies were allowed to bite the monkeys immediately after being caught, the infection in

the latter manifested itself in about two weeks; but when the flies were first fed on sleeping-sickness cases the monkeys experimented upon did not show trypanosomes in their blood until after EIGHT WEEKS. This singular fact proves very clearly that the flies, instead of drawing trypanosomes from the blood of the sleeping-sickness patients, who had very few, if any, in their peripheral circulation, actually discharged in the blood of the latter all, or most, of the parasites they were carrying.

Then, again, I do not consider that the Commissioners proved that the flies caught in the vicinity of Entebbe were carrying the trypanosomes of sleeping sickness. They only proved that the flies were carrying trypanosomes; but who can tell whether these trypanosomes were those of sleeping sickness? The Commissioners state themselves that they examined some oxen in Entebbe, and found in the blood of these animals a trypanosome, which was neither *Trypanosoma brucei* nor *Trypanosoma gambiense*. And surely many other species of trypanosomes are to be found in the blood of the mammals, birds, batrachians, and fish of Uganda.

Lastly, I do not consider that they proved that the fly is merely a passive carrier of the infection. The experiments made by Colonel Bruce on Nagana with tsetse flies (*Glossina pallidipes* and possibly *Glossina morsitans*), those made by Rogers on Surra with *Tabanidae*, those made by Theiler on "gall sickness" with *Hippoboscidae* (*Hippobosca rufipes*, *Hippobosca maculata*) prove perhaps (though I am inclined to explain them in a very different way), that a fly disturbed while sucking a *Trypanosoma*-infected animal *may* convey the disease to a healthy animal by means of its blood-soiled proboscis, just as malaria may be conveyed by a needle soiled with malaria-infected blood. But this mode of transmission does not seem to be the usual one. It does not explain why Nagana is carried exclusively by a *Glossina*, and not by any of the many other blood-sucking insects which attack horses and cattle outside the tsetse stations, or "fly-belts" as they are called. It does not explain why sleeping sickness is likewise strictly confined to the tsetse tracts, and is not propagated by the numerous blood-sucking insects and acarida which swarm in the huts of the natives or cover their bodies.

But there are many other objections against the Commissioners' experiments at Entebbe.

The monkeys used for the experiments were kept in the

open without any protection from blood-sucking diptera or any other source of infection; and this in a district in which sleeping sickness was prevalent. Some of the monkeys used for the experiments belonged to an Asiatic species (*Macacus rhesus*), and were purchased in England. The majority, however, were specimens of a local *Cercopithecus*. The Commissioners do not tell us whether any of the Uganda monkeys presented trypanosomes in their blood, or whether there was any sickness amongst these animals, under natural conditions. A careful examination of the local monkeys would have been very important. The Lokoja natives, knowing nothing of trypanosomes, assert that the tsetse extracts from a certain small red monkey the poison with which it inoculates the bush-cow or dwarf buffalo.

The flies were not reared for one or more generations in the laboratory, as should have been done, in conformity with the numerous and masterly experiments which have been carried out in connection with the intermittent fevers of man and the hæmoglobinuric fevers of cattle. They were collected at random, and hundreds of flies belonging to two or more varieties (species ?) were indiscriminately used in each experiment.

All these experiments will have to be repeated in a thoroughly scientific way. Meanwhile, I believe that the bond of association between the trypanosome of sleeping sickness and *Glossina palpalis* is far more intimate than Colonel Bruce suspects.

At the same time as the publication of the "Further Report" by Colonel Bruce, Dr. Nabarro and Captain Greig, though it was received long before, the Royal Society published several other reports by the members of the first Commission. One of these reports, by Drs. Low and Castellani, is devoted to the clinical aspects of sleeping sickness. The symptoms of the disease and its morbid anatomy, both macroscopical and microscopical, are fully and admirably described. There is one point, however, on which I cannot agree with the authors. They state that polyadenitis is a common condition amongst natives, and cannot, therefore, be regarded as a special symptom of sleeping sickness. They say that it is difficult to determine what produces this polyadenitis, but that perhaps frequent skin diseases, syphilis, and verminous invasions may be responsible. Fully admitting that syphilis and other diseases may be in Africa, as elsewhere, a fruitful cause of glandular enlargements, I believe,

nevertheless, that the glandular swellings met with in natives suffering from sleeping sickness, or living within the affected areas, are decidedly symptomatic of the trypanosome infection. An analogous polyadenitis is met with in Surra, in Nagana, in Dourine and in *Mal de caderas*. Kauthack, Durham, and Blanford, as well as Plimmer and Bradford, have laid special stress on the hypertrophy of the lymph-glands in Nagana, and especially about the region of inoculation. They have also shown that the swollen glands contain the parasites. Indeed, the trypanosomes are present in the glands from one to three days before they are discoverable in the blood, and they may be very abundant in the glands when still scanty in the blood. From time immemorial the enlargement of the cervical glands has been considered a premonitory sign of sleeping sickness by the natives of various parts of Africa affected by the disease. Winterbottom tells us that slave-traders considered these swollen glands as a symptom of sleeping sickness. "They either never buy such slaves, or get quit of them as soon as they observe any such appearances." Dr. McCarthy, Surgeon-Major Gore, and Dr. Corre, report that the natives of Senegambia believe that sleeping sickness may be cured by a timely cauterisation or extirpation of the swollen glands. Dr. Christy discovered that the area of distribution of enlarged cervical glands in equatorial East Africa agrees with that of sleeping sickness.

My rapid survey of the work which has unravelled the mysteries of sleeping sickness would be incomplete if I did not mention Mr. E. Austen's admirable monograph of the tsetse flies. Mr. Austen's book, with its careful description of the various species, with its copious information on the bionomics of these flies, with Mr. Terzi's illustrations, will certainly prove of invaluable aid in future researches.

The last step in the history of sleeping sickness is one of great importance. The lady patient examined by Sir Patrick Manson on October 3rd, 1902, and so sagaciously diagnosed as a case of *Trypanosoma* fever, began to show symptoms of sleeping sickness about two months ago, and died of the disease on November 26th, 1903—two years and three months after the presumed date of infection. At the autopsy, and subsequent microscopical examination of the brain, Drs. Mott and Low found unequivocal evidence of sleeping sickness. The history of this case proves beyond doubt that Europeans are liable to sleeping sickness; that the disease is a *Trypanosoma* infection; and that the so-called "*Trypanosoma* fever" is only a pre-

cursory stage of "sleeping sickness." The trypanosomes from this case, in its early stage, were the very ones I had compared with those found by Castellani in sleeping sickness cases and declared morphologically identical.

If sleeping sickness is a *Trypanosoma* disease, if a tsetse is the means of its dissemination, the study of the bionomics of the *Glossinæ* is the most important step with regard to prophylaxis. Mr. Austen, in his valuable Monograph, has given us a very full account of the information so far collected with regard to the life-habits of tsetse flies. I also have studied the bionomics of the *Glossinæ*, but my conclusions are somewhat different from those of Mr. Austen. Tsetse flies are confined to Africa. As a genus they have a wide range throughout the greater part of the continent, but the limits of the various species are very imperfectly known. Far more definite and important is the knowledge of their local distribution. This, however, must be considered collectively because, hitherto, writers have not distinguished between species. No doubt the habits of different species will be found to differ, and perhaps these differences will explain some of the discrepancies between various observers. But whatever the species, and whatever the part of Africa in which they have been observed, authors are unanimous as to the location of the tsetse stations. The *Glossinæ* are never found on mountains; they are seldom seen above 3000 ft.; they are absent from extensive plains, or other open places; they are rarely found in cultivated patches. Their habitat is always in the neighbourhood of water: along the banks of rivers, round the coasts of lakes, on low riverine islands, in swamps and meres, especially at the foot of mountains. They are most numerous along the water's edge; they become scarcer and scarcer as one advances inland, and disappear entirely within a few miles of the water. The places they occupy are sharply defined and permanently established. These places or stations are called "fly belts," and the natives know their limits precisely. The fly belts vary greatly in disposition and extent. Not infrequently they occur on one side of a stream, but not on the other. Along the courses of rivers, or in the low country bordering a coast line, so-called "fly belts" may extend for hundreds of miles, varying greatly in width according to the nature of the country. In such cases, however, tsetse flies are not found at every point throughout the belt, but in particular patches, the area of which may be quite small. Thus, according to Bains, "the fly is extremely local, and

extensive districts in which it prevails may be passed through by the aid of guides who know the 'patches' of fly, just as a pilot knows the shoals of an estuary" These fly patches are usually confined to reed swamps, to strips of jungle, to banana bushes, or to patches of mosani or mimosa forest. In short, the essential condition of a tsetse station are: the presence of water, a thickly-wooded district, and a loose, fragmentary soil. The nature of the soil is probably of great importance. As a rule, the fly patches are in rich sandy ridges, near marshy spots. The limitation of the tsetse to definite tracts or "belts" has given rise to much speculation. The prevalent opinion is, that the fly waits near the water to feed on the animals that come down to drink. Mr. Austen advances a new hypothesis. He says: "The limitation of the tsetse to 'belts' is not as remarkable as might at first sight appear. There can be little doubt that it is due to a characteristic social tendency of the order to which these insects belong, which, though frequently overlooked, is exhibited by the majority of species of diptera, and has attracted special attention in the case of the tsetse, owing to their blood-thirsty nature, and the fatal consequences of their bites." He further says that, in spite of the fact that the distribution of most species of diptera is remarkably wide, it will be found in the majority of cases that individuals of the same species do not occur everywhere throughout its area, but are confined to particular spots, and wherever they are met with, they are usually to be found in some numbers. I do not think we can class tsetse flies among the "social insects." I can see no purpose for the association. As to the confinement of diptera to particular spots within the area of their distribution, it is in conformity with the general laws of distribution. All animals, whether social or not, are confined to particular stations which offer the necessary œcological conditions. Besides, although Mr. Austen thinks that "the occurrence of tsetse flies in belts is not difficult of explanation," he says himself that "the fact that animals may be in perfect safety on one bank of a stream, when bushes on the other side are full of the fly, is exceedingly hard to understand." Mr. Austen, although fully aware of the peculiar distribution of tsetse flies, is not unfavourable to the theory, often propounded by travellers and sportsmen, that the fly follows the big game. I think I may safely state that the distribution of the tsetse fly is in no way connected with big game. The tsetse fly is not found in open plains,

although such plains may be literary swarming with big game; and, on the other hand, places devoid of game may be very dangerous "fly zones." The marked peculiarities shown by the distribution of tsetse flies exclude altogether the big game theory. The invariable association of the tsetse with streams, rivers, or other bodies of water, is undoubtedly a very important feature in the natural history of *Glossinæ*, and one which needs careful investigation. Two main facts determine the distribution of animals: their breeding habits and the nature of their food. In the pupiparous genus *Glossina*, the association with water can only be related to food habits. I therefore suggested in my earlier Paper that it may be related to the habit of feeding on fishes. A large proportion of the fish inhabiting the African rivers and lakes are air-breathers. There are several species of *Anabas*, three species of *Protopteros*, three species of *Ophiocephalus*, and no less than 100 species of siluroids. All these fish spend a great part of their life out of water, and even when they are in water, they are constantly obliged to ascend to the surface to breathe. They are quite incapable of supporting life by breathing water. When dry weather deprives their usual haunts of all moisture, these fish bury themselves in the mud, or migrate in large numbers to the nearest swamps. The siluroids of the *Genera clarias, Clariallabes*, and *Chaunallabes* are of special interest; they live in burrows, like rabbits, coming out towards evening in quest of food. The flesh of most of these fish is much esteemed by the natives, and in many places the women go and dig them out of their burrows. A connexion with the air-breathing fish would explain most satisfactorily the peculiar patchy distribution of the tsetse flies, and their limitation to the borders of marshes or river banks. Of course, the connexion might be indirect: that is to say, the fly might not be after the fish, but after some marsh rodent like *Aulacodus;* or after a fish-eating mammal, like *Potamogale velox*, which inhabits the Gaboon; or after an otter, such as *Lutra inunguis* or *Lutra maculicollis*, which have a wide distribution in Africa. I say this, because in South America the natives point to a large water mammal *(Hydrochoerus capibara)* as the original source of *Mal de Caderas*. Dr. Christy, who is now investigating sleeping sickness on the Congo, has promised me to elucidate this point. From what we know of the life-history of the tsetse flies, I think I may state that it will be easier to eradicate the *Glossinæ* than it is to destroy the *Culicidæ*.

Lately there has arisen some disagreement on questions of priority concerning the discovery of a *Trypanosoma* in sleeping sickness, and the suggestions of the part played by tsetse flies in the propagation of the disease. I have endeavoured to adhere very scrupulously to the exact facts, so as to avoid any quarrel. I hope, therefore, that the discussion will not lapse into a worthless controversy, but that it will lead to useful suggestions for the prevention of the deadly African scourge.

Gentlemen, a great scientific triumph has been achieved; let us bestir ourselves to do justice and honour to those who, heedless of danger, have wrought this noble work.

Discussion on Dr. Sambon's Paper.

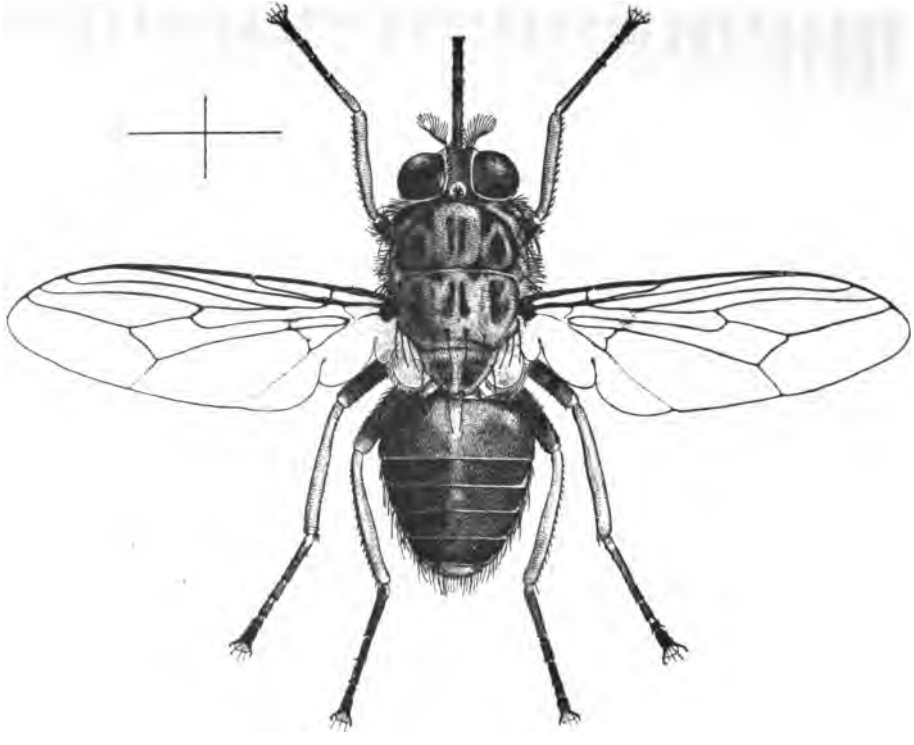
The President said he thought that Dr. Sambon's Paper, and the discussion which it was hoped would follow, would form a most valuable contribution to the *Transactions* of the Society. He considered that they had listened to a most interesting Paper, and one which would give rise to considerable discussion, in which it was hoped that a great many eminent men who had had great facilities for studying this question would take part.

He had had letters expressing regret at being absent from Professor Clifford Allbutt, Sir Michael Foster, and others; and he would now ask Sir Patrick Manson to open the discussion.

Sir Patrick Manson: I feel a great responsibility in opening this discussion, because I cannot but recognise that the subject is one of very great importance, not only to Africa—where it is really one of primary importance—but to pathology in general; for the entrance of these protozoa into the field of pathology promises to give new light in places where darkness formerly prevailed, and one must be extremely careful in expressing any opinion or view upon the subject, just when it begins to break upon the medical mind.

I was especially pleased with the last part of Dr. Sambon's Paper: that in which he expresses a hope that questions of priority will not occupy the meeting. They are always thorny questions, and they lead to a good deal of bad feeling, and do not, so far as my experience goes, conduce to general profit. Sometimes, on a Sunday, I have an opportunity of contemplating the manners and customs of a

GLOSSINA PALPALIS.



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fowl-run. My observations are usually conducted in the afternoon, between the morning meal and the afternoon meal, when the fowls are at leisure: they have picked up the last grain of the maize that has been thrown to them, and have nothing very much to do, as there is little or no chance of getting more food just then. You see in a community of fowls of that description some enterprising explorer who will wander away from the others, and by dint of perseverance, will sometimes pick up a fine worm in the corner of the fowl-run. Almost before you have observed the fact that this fowl has made a discovery all his mates have also observed the fact, and immediately this enterprising fowl is beset by hundreds of other fowls eager to share the profit, and, as a rule, the first fowl is very quickly deprived of his morsel. A second fowl succeeds in nipping a little bit off the end, a third fowl seizes it, a fourth and then a fifth, until in time you find that the discovery has become the common property of the community; with the result that I, the owner of the fowls, am the only one to benefit, inasmuch as I get probably a plumper chicken or a more nutritious egg the next morning. Now I think this is a fair simile of what happens in the field of discovery. One man gets hold of a new fact, and immediately a hundred men seek to extract a little nutriment—a little *éclat*, perhaps—from his find, and in the end the whole world benefits by the discovery. I hope these things will be borne in mind during our discussion.

I learnt a lesson from this matter of the cause of sleeping sickness. Dr. Sambon has told us already that a good many years ago I found a peculiar parasite in cases of this disease. I found, in a case that Dr. Mackenzie gave me the chance of examining, a new species of *Filaria*. I was very much interested in this subject; and finding a new parasite in what was practically a new disease, I was inclined to jump to the conclusion that the new parasite and the new disease were essentially related to each other the conclusion seemed natural.

As soon as I had made the observation, I bethought myself how I should pursue the subject, and I determined to send to all the corners of Africa for specimens of blood from the natives. I sent about 10,000 slides to Africa, and I got about 300 back; but a good many came from cases of sleeping sickness, and in every case of sleeping sickness I found *Filaria perstans*. By-and-bye, Mr. Grattan Guinness imported two cases of sleeping sickness into England for purposes of study, and was kind enough to put them at my

disposal. I was much gratified to find in the blood of these two cases also *Filaria perstans*. Now, with evidence of that sort at my disposal, I think it would have been beyond the powers of resistance of most human natures to say that *Filaria perstans* was not the cause of sleeping sickness. Yet even then I resisted the temptation. I said it was very likely: at all events, we had some ground for pursuing the subject further; and when afterwards the same parasite was found in British Guiana I was somewhat disappointed, because at that time we had no knowledge of sleeping sickness in British Guiana. I wrote to a friend there, and asked him if he knew of any cases resembling sleeping sickness in British Guiana, and he wrote back and said that a patient had been brought to him suffering from a condition of somnolence from which her friends had great difficulty in awaking her. This information somewhat encouraged the idea that, possibly in this very little-known country sleeping sickness might exist, and the existence of sleeping sickness there would further endorse the possibility as to the filaria being the cause of the disease.

By-and-bye, when Drs. Low and Castellani went out to Uganda to investigate the subject, they went out with distinct instructions from the Royal Society to ascertain as to how far this particular Filaria might be connected with sleeping sickness. Their first reports and private letters were distinctly in favour of regarding this parasite as the cause of the disease, for Drs. Low and Castellani found something like 95 per cent. (I forget the actual figures) of the natives of Uganda who were suffering from sleeping sickness to be the hosts of *Filaria perstans*. By-and-bye, on extending their observations, they made the somewhat disconcerting discovery that although in Uganda itself, or rather in the particular part they were investigating, *Filaria perstans* and sleeping sickness seemed to occur together, yet in a district where the sickness was epidemic, *Filaria perstans* seemed to be entirely absent. Thus at one stroke was destroyed a hypothesis from which I had begun to think I might derive a little credit, and I am now compelled to disavow any connection of *Filaria perstans* with sleeping sickness further than accidental.

As regards the connection of this new parasite, *Trypanosoma*, with sleeping sickness, we have to bear in mind my experience with *Filaria perstans*. My creed is—my belief is—that sleeping sickness is caused by *Trypanosoma gambiense*, but I will not say that this has been proved. My creed at one time was that sleeping sickness

was caused by *Filaria perstans*, but I could not say that it had been proved. I still think there is room for further proof on the subject of a distinct and definite connection between *Trypanosoma gambiense* and sleeping sickness. Observe that there is a difference between belief and knowledge—I believe it is the cause, I do not know for certain that it is. Lastly, the case to which Dr. Sambon has referred, a patient of mine who died recently of sleeping sickness, has added another reason for our believing in the close relationship of the parasite with the disease. But even that additional evidence is not sufficient for the scientific mind. Concurrence even in that case may have something to do with the matter.

When Dr. Castellani's discovery and Colonel Bruce's extension of that discovery were first made public, I was extremely sceptical as to the relationship of the two parasites, and for this reason : Colonel Bruce distinctly states that 28 per cent. of the whole population of Uganda have got the *Trypanosoma* in their blood. Now, sleeping sickness is a disease that certainly predisposes the human body to many kinds of parasitical invasion ; inasmuch as, in consequence of its paralysing effects on the sensory nerves, and especially on the cutaneous sensations, and the indifference to what goes on outside the patient which is characteristic of the disease, such carriers of parasites as flies, ticks, bugs, and so on, can make bites with impunity and not be brushed away. One can understand that a patient afflicted with the indifference and languor so characteristic of sleeping sickness would be extremely prone to be infected with the parasite when in this condition of physical languor, and therefore is just the person who would exhibit parasites of that character. In the same way, I believe he is specially subject to invasion of *Filaria perstans*. Therefore, it seemed to me that our evidence was nothing approaching what could be regarded as satisfactory from a scientific point of view. But when this poor lady, in whom I had found (or rather Dr. Daniels had found) the *Trypanosoma* a good many months ago—long before she exhibited symptoms of the languor of sleeping sickness (the parasite was the cause of the initial fever contracted on the Congo)—when this lady developed sleeping sickness, I thought we had a much more cogent argument than has been supplied from Uganda. She had no opportunity of being bitten while in a torpid condition, no opportunity of being bitten by *Glossina morsitans* or *palpalis*, and no opportunity of subsequent infection following the primary act of

infection, whatever it may be, that introduced the *Trypanosoma.* Therefore, I think that this case is one of very great importance, as affording, not complete but additional proof that *Trypanosoma* is the cause of the disease. Still, it is conceivable that even in her case the two conditions—*Trypanosoma* in the blood and independent disease—might concur; and, until we have obtained constant results from inoculation with pure cultures of the *Trypanosoma,* I say the subject is not closed, and we still ought to maintain an open mind as to the relations of the *Trypanosoma* and of the disease. My belief—founded on a consideration of the probabilities (nothing more)—is, that this parasite is the cause of the disease; but I cannot say that it has been scientifically and conclusively demonstrated. I hope that the cultivation experiments that have been made lately by two American scientists on the growth of *Trypanosoma lewisi* in pure culture will be applied to the decision of this question: for, until the thing is positively decided one way or another, I think one incurs a certain amount of responsibility and danger in advising anything like expensive measures with a view of counteracting *Trypanosoma* in connection with sleeping sickness. To base expensive measures on what may be only a speculation and not a fact, is somewhat dangerous ground for men responsible for the conduct of public affairs. So I would urge on those who are interested in this question to undertake these cultivation experiments. And then, if we find that pure cultures of *Trypanosoma gambiense* in an animal, or in a human subject, if anyone is sufficiently self-sacrificing to offer his own person, produce symptoms of sleeping sickness—then I should say the thing is mathematically proved, and we should be content.

I should like to point out one or two things that have struck me in connection with this matter as worth deciding. Colonel Bruce has distinctly proved that the monkey (and his experiments have been repeated in Paris and elsewhere) —or a certain species of monkey, at all events—is susceptible to *Trypanosoma* infection. This being so, is there any proof that the monkeys of Uganda have got sleeping sickness? I do not think those experiments by Bruce of injecting blood or cerebro-spinal fluid of patients infected with sleeping sickness, and subsequently inducing sleeping sickness in the monkey, along with *Trypanosoma* in the blood, are at all conclusive of the connection of *Trypanosoma* with the sleeping sickness. Supposing we assume

that this is an independent disease, and that there is no connection between *Trypanosoma* and sleeping sickness, you take the blood of a patient suffering from sleeping sickness, and having *Trypanosoma* in his blood, and you inject this into the monkey, and you produce two diseases—*Trypanosomiasis* and sleeping sickness: Suppose you had a patient suffering from plague, and at the same time from relapsing fever, and you take a little of that patient's blood and inject it into a sound man's veins: in that sound man you produce two diseases—plague and relapsing fever. You would find *Bacillus pestis*, and you would find *Spirillum obermeieri* in that man's blood; but it would not follow that the *bacillus pestis* was the cause of the relapsing fever, or that the *Spirillum obermeieri* was the cause of the plague. Neither does it follow from Bruce's experiments that a definite relationship between the *Trypanosoma* and sleeping sickness has been established.

I should very much like to know whether monkeys are subject to sleeping sickness, and whether there has been any epidemic of the disease amongst these animals.

Prof. RAY LANKESTER: May I venture, not as a member of the medical profession, but as one who takes a great interest in the subject from what I may call the natural history point of view, to make a few remarks.

My own interest in this matter began with the study, some years ago, of the *Trypanosoma* which occurs in the blood of the frog; and I believe that I was the first to study this, and to write about it, after Grubey. It had not been mentioned in the interval, and at the time (1870) I missed Grubey's account (it was a very old one), and described it as a new parasite. It subsequently turned out that this parasite was the one called *Trypanosoma* by Grubey; and since then I have always taken great interest in these parasites of the blood—rather, I should say, from the naturalist's point of view. I very much doubt, for one thing—although I see that Laveran and Mesnil take the opposite view—whether it is really right to call these parasites of the mammalia by this name *Trypanosoma*. I think zoologists would say that there is a generic distinction between the characters of the original *Trypanosoma* and the forms allied to it, which occur in mammalia and in birds; and therefore it is rather awkward that this long word has become so commonly used. I think zoologists would say that the name

"Herpetomonas," which was given by Savile Kent to Lewis's parasite (the one he got from the rat), must become that of a genus for the species of mammalian *Trypanosoma.*

May I say that I listened with great pleasure to Sir Patrick Manson's statement, which was an extraordinarily interesting account of his views and theory on the matter, and was the statement of a really scientific attitude as to the outcome of this question. He had had strong presumptive evidence with regard to *Filaria perstans*; but he did not commit himself about it; and I think that the doubt he has pointed out to us with regard to the *Trypanosoma gambinese* is absolutely correct and sound. There is a great deal of converging evidence, which amounts to cumulative evidence in favour of *Trypanosoma* being the cause of sleeping sickness, but it is not absolutely proved to be so. The possibility of another parasite accompanying the present one in the blood has to be borne in mind. We are getting to know such extraordinarily minute and varied parasites in the blood of the mammals in tropical countries, that we have to be very careful as to the conclusions which we form.

I think it is not desirable to discuss questions of priority on the present occasion; but I think that Dr. Sambon should correct the statements in his Paper by reference to the Report of the Commission of the Royal Society, in which he will find that Dr. Castellani did not attach any importance to the fact that his sleeping sickness patients had *Trypanosoma* in their blood until Colonel Bruce pointed it out; and it was Colonel Bruce who initiated and carried out the investigation which proved the invariable presence of *Trypanosoma* in sleeping sickness.

Another point which struck me was that in one or two matters Dr. Sambon seems inclined to put in some claim for himself for priority for some suggestions that he has made: a claim which I feel inclined to call in question. I refer especially to the suggestion that the *Glossina palpalis* is the carrier of the sleeping sickness *Trypanosoma.* It occurred to Dr. Sambon, as it would occur to anybody who knew the facts, that this might be so. The real interest in the matter is that Colonel Bruce found the *Glossina palpalis* where it was not previously known, and showed that its distribution coincides with that of sleeping sickness, and that it does and can carry the parasite; and he did this, of course, quite independently, and without suggestion from Dr. Sambon.

Similarly, the suggestion that a change goes on in the *Trypanosoma* whilst in the interior of the *Glossina*—that there is some further development—is a suggestion which everybody who has ever heard of a parasite undergoing a change in the body of its host will, of course, entertain. It is no use claiming priority for a platitude of that kind—the thing is to show that a change occurs. It is a possibility that observers who have *Trypanosoma* and *Glossina* under their observation are bound to entertain. But the suggestion that there is this possibility is not worth putting on record as a matter of priority—that is my opinion.

I think there is no doubt a very great deal more to be done in the study, not only of this particular *Trypanosoma* and its connection with sleeping sickness, but with regard to many other forms of the same kind. We must not be impatient. I think that a very extraordinary rapidity has been shown in the growth of our knowledge about the relation of *Trypanosoma* to sleeping sickness, when you remember that it was only the beginning of last March that 70 per cent. of the patients were found to have the *Trypanosoma* in their cerebro-spinal fluid—only last March. Well, there has not been much time lost in the investigation since then, and it is still in progress.

The PRESIDENT: I think the time has now arrived when I must consult the Society as to what they will do. We have every promise of an animated discussion on this very interesting subject; but we have nearly reached ten o'clock, and our rules will not allow us to go on after half-past. What I would submit is this: that we should adjourn the discussion—the greater part of it, with one exception, which I will name in a moment—to our next meeting on January 15th, in the hope that it will be convenient to Dr. Sambon to be with us again on that occasion; and that, in the meantime, we have the Paper and so much of the discussion as has been already contributed, together with the exception that I am about to mention, set up in type; we should then be in a position to do more justice to the subject when we return to it.

I want to make one exception. We are honoured to-night with the presence of Dr. Brumpt, who has come from Paris; and, although we should be only too pleased to have Dr. Brumpt with us again at our next meeting, it is rather too much to ask of him to postpone what he has to say to us until then. I would, therefore, suggest that we should continue for a sufficient time after ten o'clock to

hear what Dr. Brumpt may be able to tell us, and that we should then go into the next room and look at the specimens that have been prepared for us. Is it your pleasure that we adjourn the discussion, with that exception, to our next ordinary meeting on January 15th?

(The resolution was carried.)

The PRESIDENT: Then, that I understand to be carried. Dr. Sambon's Paper and the discussion which has already taken place, shall be put up in type; and may I ask those members and others who are present who desire to have copies of it, to communicate with the Honorary Secretary, Dr. Bulstrode?

Dr. E. BRUMPT: Professor Ray Lankester is quite right in saying that the tsetse fly hypothesis is of little importance, as a consequence of Castellani's discovery, when based merely on the analogy of Nagana. But there is a great difference between a mere suggestion and the arguments adduced by Dr. Sambon and myself.

Having noticed on the Congo, in December and January, 1902, that neither water, nor fish, nor manioc could be incriminated in the causation of sleeping sickness—because within the same region, whilst the inhabitants fed exactly on the same diet, but living close to a river, were decimated, those inhabiting the interior were spared; and that, on the other hand, the disease, though frequently imported to America, had not become acclimatised in that part of the world—the idea suggested itself of an intermediary host living by the water-side, and thus permitting the extension of the disease along the river margins. It is for this reason that, on hearing of Castellani's discovery, without the slightest hesitation I incriminated the tsetse flies, which alone could explain the epidemiology of the disease.

With Dr. Sambon and Sir Patrick Manson, I also consider that Colonel Bruce's experiments lack in scientific accuracy. There ought to have been control animals remaining immune after the bite of flies having fasted for a sufficiently long time, and others succumbing to the bites of infected flies. However, I consider Bruce's experiments of great interest; and were it possible to prove that the trypanosomes which developed in the monkeys were really those of sleeping sickness, Bruce would have solved one of the most important problems, especially as regards prophylaxis, which in this malady, as in many other tropical diseases, has a far greater importance than treatment.

My own researches are due to the great discovery that Castellani made while in Uganda, on behalf of the Royal Society. I think, the tsetse fly theory explains the whole epidemiology of sleeping sickness. All tsetse flies must be able to play a part in this disease, as well as in the other trypanosome diseases of Africa. These flies owe their great virulence to their voracity; to the habit of piercing the skin very frequently before settling definitely to feed; and, lastly, to the blandness of their gastric juices, which allow the trypanosomes to remain alive in their stomachs for a very long time.

The geographical distribution of *Glossina palpalis* is very wide. I found it on the River Omo, in the vicinity of Nimulé, and all along the Congo, from the sources of the Onelle to Matadi.

Sir Patrick Manson, with perfectly scientific caution, has expressed the idea that the trypanosome and sleeping sickness may possibly represent two very different morbid entities, and that by inoculating the trypanosome one inoculates at the same time the germ of sleeping sickness. The experiments I made on animals in the Congo, and those I am now carrying out in Paris, together with Professor Wurtz, dispose, I think, of Manson's objection.

A monkey (*Macacus rhesus*) was inoculated with the blood from three cases of sleeping sickness, in the abdomen and beneath the skin of the thighs, as well as with the cerebro-spinal fluid of these patients into the vertebral canal. Repeated examinations during six weeks proved negative. The animal presented neither trypanosomes nor symptoms of sleeping sickness. The same monkey inoculated with the blood of another monkey which had been successfully inoculated, presented in eight days trypanosomes, and simultaneously, symptoms of sleeping sickness.

The inoculations which remain negative as regards trypanosomes are equally negative as regards the symptoms peculiar to sleeping sickness.

ADJOURNED DISCUSSION, JANUARY 15TH, 1904.

THE PRESIDENT: We will now continue the discussion of the Paper which Dr. Sambon read before us at our last meeting, and the discussion of which was commenced but not finished. We have before us in print the full text of that Paper, and the remarks made by Sir Patrick Manson, Professor Ray Lankester, and Dr. Brumpt.

I will, in the first place, call upon Dr. Nabarro, whose name is so honourably associated with the recent researches in Uganda, to open the discussion.

DR. NABARRO: Mr. President and Gentlemen—I am sorry that I had not returned from Africa when your first meeting on this subject was held, and that I have not had the opportunity of fully reading Dr. Sambon's Paper. I do not intend to go into controversial points to-night, but perhaps I may be able to make a few remarks which will be of interest to this Society, especially as since Col. Bruce's return from Uganda we have continued the work which was started last March. When we arrived in Entebbe we were told two facts: one by Dr. Castellani—as probably you have heard—that he had examined the cerebro-spinal fluid of patients suffering from sleeping sickness, and he had found *Trypanosoma* in 5 out of 15. The second fact was communicated by Dr. Baker, of the Uganda Service, and it was that he had found the *Trypanosoma* in the blood of one of the natives of Uganda. These two facts were extremely interesting, because so very unexpected, and they at once led us to think that possibly the *Trypanosoma* had something to do with sleeping sickness; and although it would not have done to jump at once to a conclusion, it led us to carry out the investigations which we did in the subsequent months that we were in Entebbe. While Dr. Castellani was in Entebbe we carried out a number of lumbar punctures, and thereby increased his percentage from 5 out of 15 to 19 out of 34: which works out, I think, to 56 per cent. and not 70, as erroneously stated in the Royal Society's Report. During the next few weeks Dr. Baker, curiously enough, came upon four other cases of *Trypanosoma* in the blood of natives not suffering from sleeping sickness. That was very interesting, and it showed us that *Trypanosoma* was not so rare as one might have expected. Patients with the *Trypanosoma* in their blood came to the dispensary because they had an attack of fever, and really there was very little else the matter with them. We had them under observation during the whole time that we were in Entebbe; I saw all the men two months ago, before I came away, and they were all practically well and at their work. We punctured them every month: they did not mind the operation, and it did not seem to do them any harm; we always gave them chloroform, and they generally got up about fifteen or twenty minutes afterwards and went away. If you read

the Report published by the Royal Society, you will find that in the early months they had enough *Trypanosomata* to show in blood films; but later on we had to take large quantities before we could find *Trypanosoma*. This would last for several days, or perhaps a week or two, but on no occasion did they show *Trypanosoma* in the cerebro-spinal fluid—that is, in the early months. Later on, in July, two of them showed *Trypanosoma* in the cerebro-spinal fluid. Of course we were rather pleased at that, because we had then in our minds the theory that *Trypanosoma* was possibly the cause of sleeping sickness. So we looked carefully for any signs of sleeping sickness in these patients, but beyond very indefinite symptoms we could find nothing the matter with them. Therefore we examined them again in a month's time, but we could not find *Trypanosoma* present then, unfortunately. I examined them afterwards three times at intervals of a month, and there was no *Trypanosoma*. I think it is possible that when we found the *Trypanosoma* in the cerebro-spinal fluid there was an appreciable amount of blood, and that was one objection that we had to consider, namely: the likelihood of blood being mixed with the cerebro-spinal fluid; but that does not apply to a large number of cases. These patients are still under observation by Captain Greig, whom I left in Entebbe, and I am thinking that these people will still develop sleeping sickness.

Then, after Dr. Castellani left, the theory being a very suggestive one, we devoted our attention practically exclusively to the *Trypanosoma*. We did several *post-mortems*, and looked for *Streptococcus*, which was found in the heart blood and lateral ventricle fluid in some of the cases. A *Diplococcus* was found in two of the *post-mortems*, but we did not do a very large number, because the *Trypanosoma* theory seemed a suggestive one, and we devoted all our energies to that part of the subject.

The first point in that connection is the result obtained by lumbar punctures in cases of sleeping sickness. In practically every case — I cannot remember the exact number—we found *Trypanosoma* in the lumbar fluid of sleeping sickness patients. True, it is sometimes extremely scanty, and on more than one occasion we have had to repeat the lumbar puncture two, three, and four times before finding *Trypanosoma;* but on the fourth occasion we have found it present. Again, in some of them we found a large number of *Trypanosomata*. We drew off 10 c.c. of the fluid, centrifuged it, and poured off the super-

natant liquid. Sometimes a drop of the residue will show two or three in the very first field, although there might not be a single red corpuscle in twenty fields of the microscope—and that point I would emphasise. I am practically sure that the *Trypanosoma* does not get into the lumbar fluid owing to leakage of blood. In fact, I did a case of a Persian (the first known case in a non-African race), and his cerebro-spinal fluid was simply teeming with *Trypanosomata*, and yet there were no red corpuscles in twenty fields of the microscope.

I would point out that this question applies not only to Uganda proper, for Dr. Wiggins examined a large number of cases in Kavirondo, on the north-east of Victoria Nyanza; and he found *Trypanosoma* in every case of undoubted sleeping sickness.

We examined the cerebro-spinal fluid of a Persian who had the disease, and his fluid was teeming with *Trypanosomata*. I have already told you that one does not always get it on the first occasion; sometimes one has to repeat the puncture two, or even three and four times. At times there is a good deal of fluid present, and it seems that the dilution renders it rather more difficult to find *Trypanosoma*, because there is so little deposit. Another point which we have not mentioned, and which rather struck me, was this. The flow of the lumbar fluid was at times very slow and irregular; so it was suggested to start the flow by aspiration with a syringe, and one was surprised then to see the fluid spurt out. That happened on several occasions, and one managed in that way to save a good deal of time. No blood was obtained with the fluid, and so it did no harm to the patient.

Next, as to lumbar punctures in controls, that is, in patients who did not show obvious signs of sleeping sickness. It is rather difficult to get a large number of these; it is difficult to persuade a native to come and take chloroform, and submit to a lumbar puncture. I think the numbers are: 15 which we did in our laboratory at Entebbe, all with a negative result, and 7 by Dr. Wiggins, giving a total of 22. Then there were 5 with *Trypanosoma* in their blood—they were extremely interesting, because we lumbar-punctured them once a month regularly from March to July. In the case of 2 of them, they never showed the *Trypanosoma* until July. Well, that I think is very important to bear in mind, because we know that these patients had a considerable number of *Trypanosomata* in their blood, and on several occasions an appreciable deposit

of blood in the cerebro-spinal residue; and in spite of that there was no *Trypanosoma* to be seen. So that although there were a number of *Trypanosoma* in the blood, and an appreciable quantity of blood in the lumbar fluid, there were no *Trypanosomata* to be seen in the fluid itself.

Leaving the lumbar fluid, we come next to the blood of patients suffering from sleeping sickness, and the blood of controls. When I tell you that to find the *Trypanosoma* in the blood we very often had to draw off 10 c.c., and centrifuge it three or four times, you will understand that after having done about 25, we had had about as much as we found to our taste. It took quite a long time to do this, and in 15 out of 16 we found *Trypanosoma:* in the 16th, we tried seven times, but could not find it. Then we had to do controls, *i.e.*, healthy people: and this, perhaps, I had better split up into different categories. The first, that of healthy people from non-infected areas in Uganda. There are parts of Uganda, especially in the interior, where there is no sleeping sickness, and it was from one of these that these people came. They came into Entebbe to do a month's work, and ten of them used to be sent to us every day to have their blood examined. Our method was to draw off 10 c.c. and centrifuge it three or four times: we used to have three or four going at the same time, and in that way managed to get through several of them in a day.

We examined sixty-six of these people, and they were all negative. That, I submit, is a very interesting and important fact which ought to be borne in mind.

Similarly from a non-infected area in East Africa, where Captain Greig went to make some observations on *Glossinae*, fifty natives were examined, and in every case with a negative result. Therefore, you have 116 observations of healthy individuals in a non-infected area, and all showing absence of *Trypanosoma* from the blood.

I think I may say that all, except perhaps one or two, were examined by centrifuging the plasma after it had been pipetted off. This operation was repeated three times, and the residue examined again, and the *Trypanosoma* seen in the third or fourth residue.

The third category were those natives from the infected areas, viz.: the shores of the Victoria Nyanza, and especially the islands. We find that of seventy-four natives examined from these districts, *Trypanosomata* were present in 17 cases; in other words, 23 per cent. These numbers are slightly different from those given in our Report, but I think they are a little more accurate. I have altered them

a little, because they included other cases which do not come in this category: for example, you will find that, in our Report, five patients of Dr. Baker's are included in them. But they themselves were selected from a number of other natives. These—the seventy-four—were taken at random, and the *Trypanosomata* were found in seventeen of them, in other words, 23 per cent.

Then, lastly, we have these five natives, in whom they were found by accident when they had fever on them; and one European who had been exposed to the bite of the tsetse fly: he developed *Trypanosomiasis*, and was sent home.

We began to think that *Trypanosoma* had something to do with the disease, and we turned our attention to the tsetse fly. We inquired whether they had the tsetse fly in Entebbe, and we were told "No." But a few days after, we sent out into the forest, and found the tsetse fly: this showed us that we could not rely on what we were told; and after that flies were brought in by hundreds every day.

While on the subject of flies, perhaps I may say a few words about the distribution of the fly in Uganda. Very soon after finding the fly in Entebbe, we thought it would be interesting to ascertain, if possible, whether these flies were found throughout Uganda; and a circular letter was sent out to missionaries and others, to ask the natives to collect flies and send them in; and in that way large numbers of flies were sent in to us, sorted out, and a map was made, which has been published by the Royal Society, showing the distribution of the fly throughout Uganda; and it is interesting to note that in one part of Uganda, in the district of Busoga, the fly goes a long way inland. Why that is we have not been able to make out; but on the left bank of the Nile, the Uganda provinces, the tsetse fly is practically never found in the interior. Just before I left, I sent word asking the collector to send flies from the left bank of the Nile, and I did not get a single tsetse fly, although on the right bank they are found a long way down the Nile itself.

If you look at the map showing the distribution of the disease, and also at the map showing the distribution of the fly, you find a very close similitude between them, and you see that practically the distribution of the fly and the distribution of the disease are identical. I think myself that that is a very strong argument in favour of *Glossina* being the cause of dissemination of the disease. I travelled up the country, and I was struck by the fact that the fly

is extremely localised to forest areas and to the water's edge. The three most constant features in these fly areas are water, forest and dense undergrowth: I have never found them in papyrus swamps nor in grassy plains. The islands in the lake are full of the fly, and the islands are hotbeds of the disease. Some of the islands are practically depopulated, and the curious distribution of the fly certainly adds to the probability that it is the carrier or disseminator of the disease.

Then, as to experiments performed on animals. We used a large number of animals—monkeys, dogs, guinea-pigs, rats, sheep, goats, donkeys, oxen and a jackal. Unfortunately, the animal experiments were not very satisfactory. We found that only four animals took the *Trypanosoma* at all: that is, were at all affected by injections of *Trypanosoma*, viz., the monkey, the dog, the rat and the jackal. The others—sheep, goats, oxen, etc.—were not affected by *Trypanosoma* injections. They were still alive and well when I left, although they had been injected three or four times with *Trypanosoma*. The monkeys behaved very curiously. A few weeks after the injection, *Trypanosoma* appeared in the blood, but not very abundantly; it then became very numerous, but after a short time began gradually to disappear; and when I left, practically all the monkeys failed to show the *Trypanosoma* in the blood, and I am sorry to say they were all in a very good state of health. A few of them, however, died, as you have read in our Papers. One of them died of tuberculosis, and three others died after the inoculation of *Trypanosoma*, but whether as a result of that or not, it is extremely difficult to say.

The point I would like to bring out in these experiments is, that we did a number of parallel experiments. All animals were injected in pairs, the one with the *Trypanosoma* from the blood and the other with the *Trypanosoma* from the cerebro-spinal fluid, and all the blood *Trypanosomata* were taken from these five cases of what we used to call *Trypanosoma hominis*—the patients who did not show signs of sleeping sickness, although they had *Trypanosoma* in their blood. The two *Trypanosomata* behaved identically in the two sets of animals. They gave rise to similar appearances in the monkeys; behaved similarly in the dogs and the rats; and if the *Trypanosoma* did not appear in the one set of animals, it did not appear in the other. That led us to think we were dealing with one and the same *Trypanosoma*.

Other experiments favoured that view still more. When the *Trypanosoma* had disappeared from the blood in these injected monkeys, I re-injected them with the same *Trypanosoma*, to see whether that *Trypanosoma* would appear again in the blood, but it did not. The animal had apparently become to a certain extent immune. Then I thought one might see what would be the effect of injecting the other *Trypanosoma*. You see, in the first place, one had immunised the monkey with blood, or cerebro-spinal *Trypanosomata*. Then I injected the cerebro-spinal or blood *Trypanosoma;* and when I left the country the second *Trypanosoma* injected had not appeared in the peripheral blood. I think that tends to show that the two *Trypanosomata* are identical.

I come next to the feeding experiments, which have given rise to a certain amount of criticism. The flies which were used were, with very few exceptions, all fresh in from the country, and they were always kept overnight, so as to make them develop an appetite and to make sure that *Trypanosoma* would not be present in them. Then, on the next day, they were fed on an animal specially kept for the purpose: so that every fly brought into the laboratory was kept overnight, and then fed on an animal kept specially for that purpose, which was called a "fresh-fly" monkey or dog with a clean bill of health. Then the flies were kept for twenty-four or forty-eight hours, and finally put into the feeding-boxes for the feeding experiments. It has been said that we might have bred our flies, but that would have been extremely difficult. One could have done it, but seeing that we wanted a large number of flies, and that each female lays only one larva, and that that larva takes six weeks to become a fly, I am afraid that it would have taken too long to get as many as we wanted. I think we did develop about 100, or perhaps 120, young flies, in the whole eight or nine months that I was there.

Dr. Sambon, in his Paper, says that the fresh flies that we put on a monkey took only two or three weeks to give rise to *Trypanosoma* infection; whereas flies which had first fed on *Trypanosoma* patients took six or eight weeks to produce *Trypanosoma* in the monkey. That is true, but there are one or two reasons which I can give you to show why that was the case.

The fresh fly experiments were *pushed;* in other words, we used to get in about 100 flies a day, and, after keeping them overnight, we used to put them all on a monkey, so

as to get the experiments positive as soon as possible. In that way sometimes as many as 40 or 50 flies were put on a monkey, and all being very hungry they always fed very well: that explains why these experiments succeeded rather quickly. Since the hut-tax people have been kept out of Entebbe, the bites of the insects have been less certain of conveying *Trypanosoma*. I have placed as many as 750 flies on one monkey, and yet it has shown no signs of *Trypanosoma*.

Then there is another point. Dr. Sambon says that it took six or eight weeks to infect monkeys by the flies fed on sleeping-sickness patients. That is quite true. The fact is, the patients did not like the flies biting them very much. They often used to put their hands under the blankets and to take off the cages containing the flies, so that quite few flies were fed. Also, it seems that the flies got rather tired of the same patient; after feeding on him a few times, they did not feed nearly so well.

There are one or two other points on which I should like to touch. First, Dr. Sambon asked whether *Trypanosoma* is to be found in other animals. We did find *Trypanosoma* in other animals; but as we have not yet presented our Report to the Royal Society, I am not yet able to give you the details as to this, only that they were different *Trypanosomata*. It has been asked whether *Trypanosoma* has been found in healthy monkeys: but I say "No" most emphatically; we never found *Trypanosoma* in a healthy monkey, that is, a monkey straight from the field. All the monkeys were regularly examined as controls for weeks; but *Trypanosoma* never appeared except as the result of the feeding experiments or of inoculation. I am sure *Trypanosoma* is not a normal parasite in monkeys in Entebbe.

It has been suggested that *Trypanosoma* might be conveyed by some other blood-sucking insect; but the only other blood-sucking fly is the *Stomoxys*, and we did some experiments with them.

As to what Sir Patrick Manson said about the injection of blood from patients suffering from sleeping sickness, I would venture to point out to him that all the blood injections we made were from patients showing no signs of sleeping, and yet having *Trypanosoma* in the blood.

I have brought a few photographs which I think may interest the Society. They were taken by Mr. Stordy, the Government Veterinary Officer. They include a photo-

graph of the Persian patient, and one or two of the natives of Uganda.

Dr. F. W. MOTT said that his experience was pathological, not epidemiological. At present there were two theories for the epidemiology: (1) *Trypanosoma*, (2) *Diplococcus*; and Dr. Castellani had latterly adopted both, the micro-organisms being of the nature of a secondary infection. With regard to the morbid histology of the central nervous system, Dr. Mott had little or nothing to add to his original description, which was published in the *Transactions* of the Pathological Society. Examination of a number of cases from Uganda, and the European case of Sir Patrick Manson, had only shown the correctness of his presumption that the chronic inflammation of the membranes and nervous substance of the whole brain and spinal cord existed as exhibited by an extraordinary accumulation of *Lymphocytes* in the subarachnoid space, and in all the rivers, rivulets and streams of the cerebro-spinal fluid which surround the blood-vessels (the so-called Perivascular lymphatics)—channels which carry the special nutrifying fluid of the brain, and serve not only for the special metabolic processes of nutrition and the gaseous exchanges, but also to wash out the waste products. Such accumulation of *Lymphocytes* would interfere mechanically with the circulation of the fluid, and biochemically by the *Lymphocytes* taking up the oxygen which was normally intended for the nervous structures; hence a condition would arise not unlike that which he had seen in internal hydrocephalus, where ventricular accumulation of fluid produced lethargy and stupor, paresis, tremor, fits, etc. At first there was functional depression rather than destruction of the cells and fibres, later a true degeneration occurred. He might say that he had observed in several cases which he had examined, including the European case, a chemical change in the myelin sheaths without actual Wallerian degeneration, and he had observed the same appearance in certain chronic infectious processes. In the two cases which were in Charing Cross Hospital, a striking feature was the universally-enlarged lymphatic glands; and he found, on looking through Colonel Bruce's report, that in every case, with one exception, the glands were enlarged: the exception being a case of cerebral tumour and not sleeping sickness. A missionary who attended the meeting of the Pathological Society four years ago, stated that enlarged glands constituted a well-recognised symptom. They

had, then, three definite pathological facts: (1) excess of *Lymphocytes* in the blood contained in the vessels of the brain; (2) accumulation of *Lymphocytes* in the perivascular lymphatics and the subarachnoid space; and (3) enlarged lymphatic glands. In two of the cases recorded by Colonel Bruce, the glands were suppurating. Admitting that the glands of the groin and other superficial glands were very liable to enlargement in the Negro, owing to sores, etc., and admitting that syphilis was very prevalent among Negroes, still the fact remained that those causes would not account for practically nearly all cases having glandular enlargement, or for the special pathognomonic change in what constituted the special lymph canalicular system of the nervous system. Was this due to *Trypanosomiasis?* that parasite being really a blood parasite. Or was it due to an organism such as the Portuguese described, the *Diplococcus?* which, as far as he could see, was the same as Dr. Castellani had discovered, and to which in his first report he pinned his faith as the cause of the disease. Or did this organism, as he subsequently held (after his observation of the *Trypanosoma* in the cerebro-spinal fluid), act as an important subsidiary cause in a secondary infectious process? He stated in his report to the Royal Society that he had grown it with absolute certainty (and always pure) from eight out of ten *post-mortem* examinations from the cerebro-spinal fluid and blood of the heart taken with aseptic precautions. *In vita* he (Dr. Castellani) had found it only once in the blood, but he had grown it in two cases out of three from the cerebro-spinal fluid removed by lumbar puncture. In the *Journal of Tropical Medicine*, of June 1st, 1903, he had given further observations showing that that organism was present in the blood of the heart in 32 out of 39, and in 30 out of 39 in the cerebro-spinal fluid. In connection with those observations, Dr. Mott remarked that that infection would account for the lymphatic gland enlargement, and for the appearances which the cerebro-spinal, sub-arachnoid, and canalicular systems manifested in every genuine case of sleeping sickness. He had only had the opportunity of examining the lymphatic glands in two cases: one was that of the European patient of Sir Patrick Manson, and the other was an enlarged gland which he had not previously examined from one of his old cases. In both of them he found the *Diplococcus*. In the case of the former, every section of the gland showed numerous clumps of those organisms lying in the lymph channels of the gland, which were filled with cells and fibrin like the

appearances presented by the *Alveoli* in croupous pneumonia. It might be said that *Diplococcus* or *Streptococcus* infection was due to bed-sores and lung disease. He did not think so, for the following reason:—Dr. Eyre and himself had withdrawn cerebro-spinal fluid *post-mortem* from 21 cases of insane patients who had died mostly with lung disease, and many with bed-sores; and after careful examination, in not one did Dr. Eyre find *Diplococci*, and in one only *Streptococci*, and that was a case of suppurative *parotitis*. *Staphylococcus aureus* and *albus* were occasionally found, and sometimes *Bacillus coli*. He had been unable to find *Trypanosoma* in the blood-vessels or sub-arachnoid spaces of sections of the central nervous system, although he had looked at hundreds of sections. It might be urged that they could not be seen in sections, only in smears; but his assistant, Dr. George Watson, and he himself, had found that sections of the brain of a rabbit dying from Surra showed enormous numbers of the *Trypanosoma*, enough to produce mechanically the profound changes observed in the nervous system. But against the fact that they could not be seen in the vessels, it might be said that they took on a different form, or were hidden in the tissues in sleeping sickness. He did not regard the experiments on animals as conclusive. The strong arguments in favour of the *Trypanosoma* being the cause of that remarkable disease, to his mind, was the admirable research as regarded the distribution of the fly, the distribution of the disease, and its correlation with the existence of the fly and the existence of the *Trypanosoma* in the blood of the natives by Colonel Bruce and his assistants. The case under Sir Patrick Manson's care was of great importance in showing the chronic nature of the disease. That was of exceptional interest, because they found that the patient for a long time exhibited the *Trypanosoma* in the blood, and then died with the diplococcal infection, which in this instance might therefore be assumed to be related to the onset of the symptoms.

MR. JAMES CANTLIE: I should like to ask Dr. Nabarro a question about this spreading of the disease. Has this spreading of the disease ceased? Is it limited to the districts marked on the map, or does it spread in all directions? I should like to ask whether or not Dr. Nabarro has gathered that the disease is stationary, or whether it has only reached a certain stage?

DR. H. T. BULSTRODE: So far as I understand the disease, its medium of conveyance is the *Glossina palpalis;* and if this view is wholly true, the danger of contracting the disease must be limited to the areas already inhabited by the fly, or areas to which it may be carried. We have heard about the distribution of the fly in Uganda, but not in Senegambia or Angola. I should like to know whether the areas of the disease and of the fly correspond there. In his Paper, Dr. Sambon drew a terrible picture of the possible spread of the disease up the Nile, across the Suez Canal, and along the coast to India. I fail to see how that could happen. So far as I gather, the disease is not directly communicable from person to person, but only through *Glossina palpalis*, and therefore in the area of the distribution of that fly. Dr. Nabarro has spoken of the depopulation of an island by sleeping sickness. This fact suggests that the distribution of the fly has extended, or that the *Trypanosoma* may have other agencies of spread than *Glossina palpalis.* Otherwise, it is difficult to understand how the island in question became peopled in the first instance. It appears to me that if the distribution of *Glossina palpalis* is not altering, there should be little danger to the residents of fly-free areas in the importation of infected human beings, as the disease is not directly communicable. Probably, however, we are only in possession of part of the whole story.

Mr. E. E. AUSTEN, of the Natural History Museum, South Kensington, in writing to express his inability to be present at the adjourned discussion, asked permission to state that his interpretation of the "exact facts" was at variance with that of Dr. Sambon. Mr. Austen adds: "The point is a small one, but it should, I think, be cleared up. According to Dr. Sambon, Dr. Brumpt and himself were "the first to publish a definite opinion" as to the agency by which sleeping sickness was carried, and Dr. Sambon had stated that his views had been expressed "in a lecture on Sleeping Sickness delivered at the Livingstone College, and subsequently published in the *Journal of Tropical Medicine* of July 1st, 1903;" and Dr. Sambon adds: "Dr. Brumpt's observations were published on July 2nd . . . . . Brumpt incriminated *Glossina morsitans;* I suggested a West African species, and more especially the widely-distributed *Glossina palpalis.*" Now it is true that in the reprint of his former lecture, to which Dr. Sambon refers, he gives a figure of *Glossina palpalis* (copied, *without acknowledge-*

ment, from my *Monograph of the Tsetse-Flies*), but the illustration appears merely as Fig. 3, without any name or description whatever! Moreover, in the text, after indicating reasons for supposing that "a fly of the genus *Glossina*" is the carrier of the disease, and stating that "the genus *Glossina* comprises several species, some of which have a wide distribution in West Africa," Dr. Sambon simply adds: "The carrier of sleeping sickness should be sought amongst the latter." *Nowhere in the reprint of this former lecture does Dr. Sambon suggest that especial suspicion attaches to Glossina palpalis;* and, moreover, the only occasion on which he mentions this species by name is in connection with remarks on the occurrence of tsetse-flies in general in the vicinity of water, when he refers to my observations on the habits of *Glossina palpalis* in the neighbourhood of Freetown, Sierra Leone.

In his Preface to my *Monograph of the Tsetse Flies*, after alluding to my determination of ten specimens of *Glossina palpalis* among a small box of biting flies just received from Entebbe, Uganda, and sent home by Colonel Bruce, Prof. Ray Lankester wrote as follows: "This particular species of tsetse-fly is essentalliy a West African species, known from the Gambia to the Congo. It certainly suggests the need for an inquiry into the possible connection between this fly and the sleeping sickness, when we remember that that disease has been established for years on the West Coast of Africa, but was unknown in Uganda until two years ago. Sir Henry Stanley met with "tsetse-fly" for a long distance on the Upper Congo; and it is suggested by Mr. Austen that *Glossina palpalis* reaches Uganda by way of the valleys of the Congo and the Aruwimi."

Whether *Glossina palpalis* is, like sleeping sickness itself, a recent importation into Uganda, or whether—as seems more probable—the fly has been established there from time immemorial, the foregoing quotation is, I think, sufficient to dispose of Dr. Sambon's contention that he was the first to suggest a connection between *Glossina palpalis* and sleeping sickness. Professor Lankester's Preface was written on May 15th, 1903, and my *Monograph*, in which it appears, was published before the end of the following month. Dr. Sambon's lecture, in which, as I have shown, there is no reference whatever to *Glossina palpalis* in connection with sleeping sickness, only appeared in print in the number of the *Journal of Tropical Medicine*, dated July 1st, 1903.

DR. NABARRO: In reply to Dr. Cantlie, I think I may say the disease is spreading along both shores of the lake. It is spreading on the western shore of the lake, and also on the eastern shore; I understand, in both cases, approaching German territory. The Germans are becoming rather anxious about it, and they sent one of their colonial surgeons over to Entebbe, to study the disease and learn something about it. We showed him many things, and gave him a good deal of information; and he went back quite satisfied with our statements, and ready to deal with the disease, if it should spread into their territory. It is certainly spreading on the Kavirondo side, on the north-eastern shores of the lake.

With regard to how the disease is spread, that, I must own, is a difficult question, because the *Trypanosoma* is very scanty in the blood of the patients. I personally think, from what I have seen, that they are more numerous in the blood in the early stages of the disease than they are in the later stages. I have thought that possibly the fly gets a larger number of *Trypanosoma* in him in the early stages of the disease than later on. Then the period of incubation may extend over years, in all probability. There are cases on record in which the latent period extended over several years.

Again, I think the fly has been in the country for a long time, but that the *Trypanosoma* has only recently been introduced into the country; and I know for a fact that the Albert Nyanza, situated to the north-west of Victoria Nyanza, has the fly all round its shores, but there is no sleeping sickness there. That we ascertained only shortly before I came away. One of the collectors in the Uganda Service was sent there, and he found a number of flies, but there was no sleeping sickness; and one fears that if infected patients should get there, they might spread the disease. When infected patients get into the interior of Uganda the disease dies out with them, just as malaria would if there were no mosquitoes. People have gone from the shores to the interior, but the disease has never spread from them to other inhabitants of the house or other people in the village. All the *Trypanosomata* we found in animals seemed to be different from the *Trypanosomata* in sleeping sickness. I would like to remind you that the work of investigation is still going on, and that further experiments have yet to be made. There is no doubt, however, about the ravages of the disease. I believe, in Usoga itself, nearly 50,000 people died last year. It

is a perfect abode of the dead: all the villages are depopulated.

DR. C. F. HARFORD: Mr. President and Gentlemen,—I really have no right to say anything in this matter, since I have not had an opportunity of making any recent observations of sleeping sickness; but as I had a case under somewhat prolonged observation now some fourteen years ago, I may say a word or two with regard to the subject. My attention was drawn to the case by the natives, who seemed to have a great knowledge of the disease, although it was in a part of Africa where only sporadic cases occur—on the Upper Niger, at the confluence of the Upper Niger with the Benue. It seems curious that they should have so close an acquaintance with the disease, seeing that so few cases occurred, and those not as an epidemic.

One point which always impressed itself upon the natives—that is, the intelligent natives—whom I spoke to, was the occurrence of these enlarged glands. I think that is a matter of considerable importance: the natives there had distinctly the idea that if the lymphatic glands were removed, the disease would be cured: they told me that they had performed the operation, with the result that the disease was cured. My idea of that suggestion was that they probably regarded all cases of enlarged glands as sleeping sickness,; and that when they had scratched out the glands, and sleeping sickness did not develop, they thought they had cured the disease.

I was very much interested in this case, and I made certain inquiries from educated natives; and one fact to which they drew my attention was, that it only attacked the natives of tropical countries: in other words, they regarded it as a disease of the negro race.

I am interested in the subject also from the practical point of view. I have at my college students—missionaries —who have had a great deal to do with the disease, both on the Congo and in Uganda, and seen a good deal of it there. Also, in my position as Physician to the Church Missionary Society, I hear a good deal of it from missionaries who have come there, and from my friends, Drs. A. R. & J. H. Cook, who, I am sure, have contributed something to the elucidation of this problem. Although these gentlemen have not now much opportunity for scientific research, owing to their arduous medical missionary labours, still they have done a good deal, as you may have

seen from Dr. Cook's paper in *Journal of Tropical Medicine* some time ago. Naturally, he makes no mention of *Trypanosoma;* his investigations being devoted to the inquiry with regard to *Filaria perstans.*

I think we owe a great deal to Dr. Sambon for having read to us such a careful Paper, and such a large amount of evidence with regard to this important disease: and I hope that we may express the desire that some practical steps shall be taken to deal with this subject. It seems to me that whatever may be the possible conclusion to this subject, there cannot be much question that the tsetse fly is an insect which is a terrible scourge, not only to men but to animals, and that every effort ought to be made to see what can be done to deal with the tsetse fly in its haunts. I am not in any way a naturalist; but I do hope that something may be done to thoroughly go into this question.

Perhaps I may mention one practical step that has been taken in Uganda: that is, the authorities of the Uganda Industrial Mission have felt so strongly the danger of having their establishment in a fly zone, that they have actually taken the step of abandoning most valuable premises in order to get away from that district. That shows that there is a good deal of belief in the importance of this discovery. I think we ought to take steps, in order to make practical use of it.

Sir Patrick Manson, K.C.B., F.R.S.: I should like to make one or two suggestions for further inquiry. Dr. Harford spoke about sporadic cases of sleeping sickness. There is no question about the existence of these cases, for you get them in many places—on the West Coast of Africa, on the Gambia, in Sierra Leone, up the Niger—in all these districts you get occasional cases of sleeping sickness, and yet you do not get it in epidemic form. It would be interesting to know the relation these cases bear to the presence of *Trypanosoma* in the general population of these particular places.

A good many years ago I was asked to see a patient in consultation at St. Thomas' Home: a missionary who had come from German East Africa. He presented a curious combination of symptoms, and I confess I could not understand them. He had a peculiar enlargement of the glands, great prostration, and half a dozen other symptoms, which now I know must have belonged to *Trypanosomiasis.* This missionary, I believe, had never left German East Africa. If there be one case of *Trypanosoma* disease in German

East Africa, why not many? He must have been infected in some manner. If there was sleeping sickness in German East Africa, I think it is quite possible that you may have sleeping sickness in many other places; but the disease has not been recognised. This remark is borne out by what we now know about a similar disease: it has been ascertained only within the last few weeks as regards this disease that it belongs to the same category as *Trypanosomiasis*—I allude to the pest known in India as "Kala-azar," which is now known probably to be attributable to a *Trypanosoma*-like parasite inhabiting the spleen, liver, and bone marrow. It was at one time supposed that this disease was confined to one part of the country; but of late it has been found all over the country, and has depopulated some of the places through which it passed. It now appears that a similar disease exists in China. Only lately a German pathologist has recorded a case in the person of an individual who came from Pekin. I myself saw a similar case in a gentleman from South Africa; and I am fairly certain I have seen a similar case in a soldier from Aden. And this *Trypanosoma*-like disease has the same distribution characteristics that are now fairly recognised as belonging to *Trypanosoma* disease. You get places where it becomes epidemic, and places in which it is sporadic. It would be interesting to find out the exact conditions which determine the epidemicity, and (to coin a word) the sporadicity of the disease.

As Dr. Mott has suggested, I think there is probably a combination of causes in sleeping sickness, viz.: *Diplococcus* and *Trypanosoma*; and I think the experiment which he suggests, that a monkey which has *Trypanosoma* should be inoculated with *Diplococcus*, in order to see whether the double infection would produce the disease, would be a very useful one. I strongly advocate that the Commissioners now in Uganda should follow out Dr. Mott's suggestion.

I would like to make another observation in connection with the suggestion that the presence of *Trypanosoma* in the cerebro-spinal fluid was the determining cause of the symptoms of sleeping sickness. I do not hold with that idea: I think it is a little far-fetched. I think the *Trypanosoma* is the cause of the sleeping sickness; but to dogmatise as to the processes, or as to the manner in which it causes it, is going a little beyond the facts. When a patient is afflicted with inflammation of the central nervous system, naturally the parts are congested; the vessels are

congested and prone to rupture, and there is an escape of blood undoubtedly into the cerebro-spinal fluid; and one would naturally expect that any organism such as *Trypanosoma*, which is capable of defending itself against the *Phagocytes*, would remain in the cerebro-spinal fluid for some time.

I think it is necessary to be cautious in interpreting a condition of this sort. There is a certain phenomenon which occurs in connection with *Filaria*. You sometimes get rupture of the lymphatic vessels, and effusion of lymph into the tunica vaginalis, and the production of the disease which used to be known as Chylocele. If you examine the fluid in these cases you will find millions of living *Filariae*, much more abundantly than in the blood even; the reason for that being this: the ruptured vessel permits the lymph to escape and the *Filaria* with it; but the *Filaria*, not being able to find their way back, are imprisoned as fish in a net. I believe exactly the same thing happens in the case of *Trypanosoma*: that a rupture of the vessels allows them to escape. Moreover, the red-blood corpuscles would tend to settle down, whereas *Trypanosoma*, being endowed with powers of motion, would be likely to move about and become diffused. I think we should not attach too much importance to the appearance of *Trypanosoma* in the cerebro-spinal fluid, as being part of the pathology of the disease.

One word in conclusion. I think the profession has been a little unjust—I don't want to say disagreeable things, but I think the profession has been a little unjust, in the way in which it has ignored the work of the early investigators into this Uganda disease. When Dr. Low, Dr. Castellani, and Dr. Christy were sent out by the Royal Society, they got specific instructions to investigate on certain lines: more especially they were asked to determine whether *Filaria perstans* was the cause of sleeping sickness; and the plan by which they were to set about it was to see whether the areas of the parasite and the disease corresponded. That piece of work they carried out thoroughly, and they got no credit. Also, they were to find out, or endeavour to find out, any micro-organism that might possibly be considered as the cause of the disease. That they certainly did, for Castellani, in the first place, determined the presence of this *Diplococcus* which, as Dr. Mott says, is often found in such cases, and in the second place, he (Castellani) demonstrated the presence, in a large proportion of cases, of *Trypanosoma* in the blood.

Also, Dr. Christy has shown that the range of *Filaria perstans* and of sleeping sickness do not correspond. I think those early investigators have done their work thoroughly, and I think they should receive due credit for what they have done.

DR. FREDERICK MOTT: I should just like to say that I have only found this *Diplococcus* in this case of Sir Patrick Manson's, and I think he perhaps misunderstood me. I was referring to the work of Dr. Castellani and Colonel Bruce.

MAJOR MCCULLOCH: There is one point on which I should like to ask a question. Dr. Sambon says that "a fly disturbed while sucking a *Trypanosoma*-infected animal *may* convey the disease to a healthy animal by means of its blood-soiled proboscis, just as malaria may be conveyed by a needle soiled with malaria-infected blood." But I think Dr. Nabarro examined the probosces of a considerable number of flies, and actually found *Trypanosomata* in them. There is also one little inaccuracy, I think, and that is the conveying of malaria-infected blood. Of course, he can scarcely mean that malaria-infected blood can convey malaria to the human body.

THE PRESIDENT: Before calling upon Dr. Sambon to reply, I should like to tender to him our grateful thanks for his very interesting Paper, which has led to so instructive and valuable a discussion. This proposal was warmly seconded by acclamation.

DR. SAMBON: Before replying to the various criticisms, I must thank you for the kind way in which you have listened to my Paper. I am very pleased that Dr. Nabarro is here to-night; and although I take objection to some of his experiments, and especially to the conclusions he draws from them, I feel great regard for the work he has so actively carried out, together with Colonel Bruce and Captain Craig, in Uganda.

Dr. Nabarro admits the correctness of my remarks as to the time elapsing before the appearance of trypanosomes in the blood of monkeys submitted to the bite of tsetse flies; he owns that a much longer period elapsed when the flies were previously fed on cases of sleeping sickness than when they were made to bite the monkeys directly and immediately after being caught; but he endeavours to explain thi fact by stating that the experiments made with flies

not previously fed on cases of sleeping sickness were *pushed:* that is to say, that in these experiments a much larger number of flies were used in order to obtain a positive result as soon as possible; whilst in the other set of experiments not only *fewer flies were used,* but many of them were *not properly infected,* partly because the patients on which they were placed prevented them biting, and partly because the flies got tired of the same patient after feeding on him a few times. These statements now made by Dr. Nabarro are at variance with those published in the "Further Report."

The data published in the Report are as follows:—

First Set of Experiments.

Five monkeys (Exp. 114, 115, 99, 97, and 116) were submitted to the bite of flies fed previously on cases of sleeping sickness.

Exp. 114.—880 flies were employed. Of these, 522 actually fed on the patient, and were placed on a monkey after a fast of 8 hours. The monkey showed trypanosomes in its blood 65 days after first bite.

Exp. 115.—881 flies used; 509 fed, and were allowed to bite monkey after a fast of 8 hours. Trypanosomes appeared after 75 days.

Exp. 99.—582 flies used; 508 fed, and were placed on monkey after a fast of 24 hours. Trypanosomes appeared after 70 days.

Exp. 97.—294 flies used; 255 fed, and were allowed to bite monkey after a fast of 48 hours. Trypanosomes appeared after 48 days.

Exp. 116.—354 flies used; 277 fed, and were allowed to bite monkey after a fast of 48 hours. Trypanosomes appeared after 65 days.

Second Set of Experiments.

Three monkeys (Exp. 94, 130, and 131) were submitted to the bite of flies freshly caught, and not previously fed on patients suffering from sleeping sickness.

Exp. 94.—186 flies used. Trypanosomes appeared after 14 days.

Exp. 130.—1,034 flies used. Trypanosomes appeared after 29 days.

Exp. 131.—757 flies used. Trypanosomes appeared after 23 days.

In the face of these data, Dr. Nabarro's explanations are inadmissible. Indeed, you will notice that in Exp. 94, when fewest freshly-caught flies were used (186), a positive result was obtained in the shortest time (fourteen days); and that in Exp. 115, when as many as 881 flies were

used eight hours after feeding on a sleeping sickness patient, it took the longest time (seventy-five days) to obtain a positive result.

Dr. Nabarro's remarks as to the number of flies which did actually bite the sleeping sickness patients are likewise contradicted by the following statement in the "Further Report," p. 56, Section 12:—

"The sleeping sickness patients did not seem to feel the bites of the flies, as they made no complaint or other signs of inconvenience. There was, as a rule, about thirty flies in each cage, *but only those which filled themselves were reckoned as having fed.*"

My criticism of the experiments made by Colonel Bruce, Dr. Nabarro, and Captain Craig, is based on the facts published by these gentlemen in their "Further Report." I never thought of doubting their accuracy, but unfortunately Dr. Nabarro's statements to-night do not tally with those published in the Report.

Dr. Nabarro remarked that the rearing of flies for the experiments would have involved too much time. Certainly, it would have taken more time, and would have needed more care; but the results of the experiments would have been of a more definite and valuable kind. Dr. Nabarro says: "I think we did develop about 100, or perhaps 120, young flies." It is a great pity he did not use these flies. It is likewise to be regretted that Dr. Nabarro and his collaborators did not try experiments with *Stomoxys*, because some observers believe that these flies are capable of conveying the trypanosomes of nagana. Most of all I regret that—knowing that tsetse flies were to be found on one bank of the Nile and not on the other—Dr. Nabarro did not thoroughly investigate this all-important point.

Dr. Bulstrode has asked whether the distributional areas of *Glossina palpalis* and of sleeping sickness correspond in Senegambia or Angola. As to Senegambia and Angola, the available information is rather scanty, but so far as it goes it is certainly in favour of correspondence. On the Congo, Dr. Brumpt has made very careful investigations, and he has shown that there is a very close connection between tsetse flies and sleeping sickness. Dr. Brumpt noticed that where the men remained inland for agricultural work, the disease attacked the women who went to the river to get water. In other cases, where the men went to fish, they contracted the disease, while the women who remained in their villages were spared. The Trappist Fathers have a

mission at Banamia, about twenty minutes from the Congo. A few years ago, some 3,000 Lolo fishermen lived on the river bank; now this number has fallen below 300. The others have been destroyed by the sleeping sickness. Close to the mission is a village of cultivators; the latter rarely go to the river and drink the water of local springs. The disease is almost unknown to them.

Dr. Bulstrode seems to think that I have drawn a very terrible picture of the possible spread of the disease up the Nile, and by means of the coast to India. He says he fails to see how that could happen. Well, a few years ago, no one would have believed that sleeping sickness might spread from its old endemic centres on the Congo to the Victoria Nyanza; now it is rapidly encircling the lake. As to the Nile, already some doubtful cases have been described from Korosko; and we know that *Glossina palpalis* and other tsetse flies are to be found on its banks. The spread of the disease does not depend only on the distribution of its insect host, but on a number of other œcological conditions as yet unknown to us. Wherever sleeping sickness and tsetse flies coexist, their topographical distribution is the same, but geographically their distributional areas do not overlap. There are plenty of places in Africa abounding with *Glossina palpalis* and other tsetse flies, but without sleeping sickness; just as there are many places in Europe abounding with *Anopheles maculipennis* and other anophelina, but without malaria. However, in a general way, it is quite reasonable to surmise that sleeping sickness may invade any region inhabited by its necessary carrier.

Major McCulloch remarked that I was inaccurate in stating that malaria might exceptionally be conveyed from one man to another by means of a needle soiled with malaria-infested blood. But I will mention a very striking case which happened in the Hospital of St. Spirito, in Rome. While examining a slide of blood from a case of sub-tertian fever, Dr. Pinachi broke the slide and cut his finger. He washed the wound very carefully, and thought no more about it. However, after a short period of incubation, Dr. Pinachi suffered from a severe attack of sub-tertian fever; and, notwithstanding a prompt and plentiful administration of quinine, came very near losing his life.

Professor Lankester suggested that I should correct a statement in my Paper with reference to the number of cases examined by Dr. Castellani, and as to the interpretation given by the latter to the presence of trypanosomes in

these cases. I am well aware that Professor Lankester contends that Dr. Castellani did not understand that the trypanosomes found in the cerebro-spinal fluid of sleeping-sickness cases could have anything to do with the disease, until Colonel Bruce pointed it out to him. I know also that numerous articles have appeared, both in medical and in non-medical papers, expanding on Dr. Castellani's discovery of a *Streptococcus* in sleeping sickness, and even going the length of reproducing a factitious illustration of the bacterium, in order to disconnect his name from the discovery of the trypanosome; but all this is very unjust. I have nothing to correct; indeed, my statement is couched the very words employed by Colonel Bruce in his progress Report:—

"Dr. Castellani informed us of the work he had done: one especially interesting observation being that he had discovered trypanosomes in the cerebro-spinal fluid in 5 out of 15 cases of sleeping sickness. Dr. Castellani remained in Entebbe for three weeks after the arrival of the new commission, and during this time *he examined* 29 *further cases for trypanosomes, with the result that* 70 *per cent. were found to contain these parasites. Dr. Castellani, we presume, has already published these results.* After his departure, the commission continued to pursue this line of work."

In his "Further Report," Colonel Bruce says: "At the time of the arrival of the commission, Castellani did not consider that this *Trypanosoma* had any causal relationship to the disease, but thought that it was an accidental concomitant, like *Filaria perstans*."

However, he adds: "This most interesting discovery of Dr. Castellani's, which was due to his introduction of the method of centrifugeing the cerebro-spinal fluid in his search for his *Streptococcus*, has been of the utmost possible value to the present commission. It put them at once on the right track, and led to the rapid and easy elucidation of the etiology of this hitherto mysterious disease. Without a knowledge of his observation, they might have worked for months in the dark; and, in truth, they might even have returned to England still ignorant as to the true cause of the disease."

Let us place ourselves for a moment in Dr. Castellani's position. When he first discovered *Trypanosoma* in the cerebro-spinal fluid of a patient suffering from sleeping sickness, should we have jumped at once to the conclusion that the *Trypanosoma* found in this single case was un-

doubtedly the cause of the disease? Later, when Dr. Castellani found the *Trypanosoma* in 5 out of 15 cases, would it have been reasonable to incriminate this parasite, when it was known that *Trypanosoma* fever—then believed to be a distinct disease—was present in Uganda? But when the *Trypanosoma* was found in over 70 per cent. of sleeping-sickness cases, and not in the cerebro-spinal fluid of *Trypanosoma*-fever cases, and never in healthy persons, was it necessary for anyone to point out that *there might be a connection between the Trypanosoma and sleeping sickness?*

Professor Lankester then made some disparaging remarks with reference to my own researches concerning the transmission of sleeping sickness. He said there was nothing in them. The suggestion that a tsetse fly might be the carrier would occur to anybody. The real interest in the matter is, that Colonel Bruce found *Glossina palpalis* where it was not previously known. I have fully explained in my Paper the reasons which led me to incriminate a West African tsetse fly, and more especially the widely-distributed *Glossina palpalis*. I will not go over the same ground twice. I know full well that the suggestion of a tsetse fly occurred to many: it occurred to Professor Blanchard, to Professor Laveran, to Dr. Mesnil, to Dr. Brumpt, to Colonel Bruce, to Professor Lankester, and to many others; but, as Dr. Brumpt remarked, in the course of the discussion, there is some difference between a mere suggestion, based on analogy, and the deduction from carefully-observed facts. As to the discovery of tsetse flies in Uganda, surely it should not be my place to inform Professor Lankester that tsetse flies have been known to occur in Uganda many years before their "discovery" by Colonel Bruce. Sir Harry Johnston, in his admirable book on the Uganda Protectorate, says: "Flies of the genus *Glossina* exist in the Uganda Protectorate. They have been caught there by Mr. Jackson and myself, not to mention many other collectors." Professor Lankester's generosity towards Colonel Bruce is unbounded. In the preface to Mr. Austen's *Monograph of the Tsetse Flies*, he states that the discovery by Colonel Bruce of a *Trypanosoma* in nagana led to the discovery of a similar parasite in surra. Professor Lankester should correct this statement, because the *Trypanosoma* which gives rise to surra was discovered by Griffith Evans fifteen years previously; and, indeed, it was this discovery which led to the elucidation of nagana, a disease very probably identical with surra.

Professor Lankester's remarks have been followed up by a letter by Mr. Austen, also, of the Natural History Museum, in which the priority in suggesting that *Glossina palpalis* is the carrier of sleeping sickness is attributed to Professor Lankester. Evidently, the matter of priority is not so unimportant as Professor Lankester stated when discussing my claims. According to Mr. Austen, the priority is due to Professor Lankester, because the latter made the suggestion in a preface dated May 15th, 1903, but not published until the end of June. My Paper appeared in the *Journal of Tropical Medicine* on July 1st, 1903, but was read at the Livingstone College at the end of May. This as regards dates. But let us examine the reasons which led Professor Lankester to suggest a possible connection between *Glossina palpalis* and sleeping sickness. On the 15th of May, 1903, Professor Lankester received a small box from Entebbe, containing some biting flies sent home by Colonel Bruce for determination. Mr. Austen examined these insects, and found amongst them ten specimens of *Glossina palpalis*. As this particular species had not been hitherto described from East Africa, Mr. Austen surmised that it might be a recent importation into Uganda, and that it reached the Protectorate by way of the valleys of the Congo and the Aruwimi.

It is entirely on this assumption that Professor Lankester bases his suggestion. He says: "This particular species of tsetse fly is essentially a West African species, known from the Gambia to the Congo, It certainly suggests the need for an inquiry into the possible connection between this fly and the sleeping sickness, when we remember that that disease has been established for years on the West Coast of Africa, but was unknown in Uganda until two years ago." Unfortunately, Mr. Austen's assumption is erroneous. *Glossina palpalis* is not of recent introduction into Uganda. Although widely spread in West Africa, it is by no means limited to that part of the Continent. Sir John Kirk found it on the Zambesi in 1860, and Dr. Brumpt found it on the River Omo; on the Nile, in the vicinity of Nimulé; and all along the Congo, from the sources of the Welle to Matadi. However, apart from this, Professor Lankester's suggestion is very vague, and does not differ from that made previously by Professor Blanchard and others, with the exception that it limits to *Glossina palpalis* a *rôle* which is probably played by other tsetse flies.

Mr. Austen endeavours to prove that I did not suggest that especial suspicion attaches to *Glossina palpalis*. He says: "Now it is true that in the reprint of his former

lecture, to which Dr. Sambon refers, he gives a figure of *Glossina palpalis*, but the illustration appears merely as Fig. 3, without any name or description whatever!" The fact that I gave a figure of *Glossina palpalis* should be sufficient evidence that I did especially incriminate this species. At the same time, I did not wish to exclude all other species. This can be done only by means of appropriate experiments; and that is why I did not mention only *Glossina palpalis*, but said that the carrier of sleeping sickness should be sought amongst those species of the genus *Glossina* which have a wide distribution in West Africa.

Professor Lankester incriminated *Glossina palpalis* simply because he received a box of flies from Entebbe containing specimens of this species. I incriminated *Glossina palpalis* on account of other reasons already stated, and without knowing for certain that this species was to be found in Uganda. Recent information shows, I think, that I was working on sound lines. The omission of the descriptions under the figures was an oversight in the printing. It was corrected soon after in the reprints of my Paper.

Colonel Bruce and Dr. Nabarro did not determine the flies they used in their experiments; they merely tell us that they noticed two varieties of tsetse flies, one darker than the other. Therefore, even admitting that they succeeded in transmitting Castellani's *Trypanosoma* from sleeping-sickness patients to monkeys, it would be impossible to say quite definitely whether *Glossina palpalis* is a carrier, and whether it is the only carrier, of sleeping sickness.

Professor Lankester finally attacked my contention that the tsetse fly does not carry the trypanosomes in a passive way, as maintained by Colonel Bruce, but that it acts the part of a true alternative host. He says: "It is no use claiming priority for a thing of that kind; the thing is to show that a change occurs." Well, I think that I have produced a certain amount of evidence. I have stated that we find various forms of trypanosomes in the blood, and that some of these forms, such as the large, stumpy forms, with short flagellum and numerous dark-staining granules in their plasma, are undoubtedly sexual forms which must go through further development; but even if the trypanosomes underwent no change within the body of the fly, comparable to that of the malaria parasites, still I think we should have enough evidence to prove that the tsetse fly does not act merely as a passive carrier but as a true

and necessary host. Finally, the results obtained by Colonel Bruce and Dr. Nabarro in their experiments greatly strengthen my belief that the trypanosomes can be transmitted by the tsetse fly to its progeny; and that, therefore, some tsetse flies may be able to transmit the infection on issuing from their puparium.

Dr. Mott's remarks concerning the *Streptococcus* are very interesting, and tend to support Castellani's theory that both the *Trypanosoma* and the *Streptococcus* may be concerned in the causation of sleeping sickness. The bacterium might possibly be the cause of the terminal nervous symptoms, and the trypanosome its carrier from one anatomical region to another: just as *Dysenteriae amœba* may be the carrier of *Streptococci* into the liver. This association would explain the very variable length of time elapsing in different cases between the moment of infection and the onset of nervous symptoms. At one time it was generally believed that only one disease at a time could develop in man, and that only one disease at a time could prevail in a place. Now, we are beginning to understand that the morbid state may be the resultant of several disease agents; and, indeed, amongst pathogenetic organisms there may be a kind of *metabiosis* similar to that which brings about the fermentation of wines and the so-called "ripening" of cheeses.

Sir Patrick Manson expressed a doubt that the *Trypanosoma* might not have any connection with sleeping sickness. Considering that Sir Patrick Manson has himself contributed the most convincing evidence in favour of a causal connection between the *Trypanosoma* and the disease, I believe his doubt is more figurative than otherwise. The analogy between the symptoms of sleeping sickness and those peculiar to nagana, surra, and other *Trypanosoma* diseases of horses, dogs, and cattle, the constant presence of trypanosomes in patients suffering from the disease, the appearance of nervous symptoms in concomitance with the invasion of the central nervous system by the parasite, the development of sleeping sickness in people far and long removed from the endemic centres of the disease, the demonstration here in England of trypanosomes in the blood of Sir Patrick Manson's case long before the manifestation of nervous symptoms, and the striking experiments mentioned by Dr. Brumpt, show that there can be no further doubt as to a causal connexion between Castellani's trypanosome and sleeping sickness.

ON THE ETIOLOGY OF SCURVY.

By MYER COPLANS, M.B., D.P.H., Civil Surgeon attached R.A.M.C., South Africa.

(*Read: February 19th*, 1904.)

As I have not dealt at all fully with the clinical aspect of the cases referred to in this Paper, I may, perhaps, state that the cases conformed in every particular to what, I take it, would be regarded as typical scurvy. As to this no question has been raised; but I make the statement with the view of clearing up any doubts which may arise on this point.

Briefly, these cases of scurvy presented during life spongy bleeding gums, lassitude, anæmia, breathlessness on exertion, followed later on by lameness associated with synoritis in the joints, and hæmorrhages with brawny indurated swellings in one or both of the lower limbs. The urine was slightly acid. The blood showed marked poikilocytosis in the red discs. *There were no nervous lesions.* Death took place from cardiac failure usually, and *post-mortem* the blood was fluid; there were subserous, submucous, subpleural hæmorrhages. There was excess of serous fluid in the pericardium. The heart was thin and pale, and the right side dilated. The body was well nourished.

Further, I wish to explain my attitude in the matter. I have approached the subject with a perfectly open mind, and before this Transvaal experience I had no special acquaintance with scurvy. After studying this outbreak, I endeavoured to explain it by the current views as to the causation of the disease, but failed. I am therefore venturing to submit this thesis to the judgment of the Society.

I would ask you to approach the subject *de novo*—with open minds—setting aside for the present all historical writings and sayings on the matter, treating this outbreak of the disease as an entity; and in particular bearing in mind the fact that the question of the anti-scorbutic value of any food is not being raised.

I propose to lay before you some evidence pointing to the fact that the condition known as scurvy in adults is not

brought about by the absence of any particular kind of food from the dietary, but is, more probably, a specific infection of bacterial origin.

Towards the close of the Boer War, in May, 1902, there was presented the unusual spectacle in the Transvaal and Orange River colonies, of lands devastated by fire, devoid of farms, of crops, and of cattle, with the entire human population—natives and Europeans, combatants and non-combatants—gathered together into a series of small communities or units, of fixed location and definite occupation, under supreme military control. Each of these units was supplied by the military authorities with food, this food being mainly imported from over-sea, and transported up-country along closely-guarded railway tracks.

Thus every individual in every community was definitely rationed. Except in the few towns that still remained undestroyed, it was found impossible to obtain food of any description except that imported. For the inhabitants remaining in these undestroyed towns there were provision stores, which supplied goods at fixed prices, and in definite quantities only. The shops, the inhabitants, and their houses were under close military supervision by day and night.

Solely for the use of the military classes there were instituted the Field-force canteens, which supplied additional articles of food on payment. These canteens, though private ventures, were also under military control; and the demand from them by the troops for goods was nearly always in excess of their power to supply. The goods sold at these canteens were principally imported preserved foods; and thus all in British military employ were enabled in some measure to supplement their regular rations.

All other classes were dependent entirely on the Government for rations; and at this time every individual in the country could, with some degree of certainty, be accounted for officially as to dwelling-place, employment, and amount and nature of food consumed. From the military organisation of affairs, and division of the inhabitants into units, no individual non-combatant could fare better or worse in his food-supply than the remaining members of his own unit.

Thus all burgher camp inhabitants participated equally in the general food-supply for the burgher camp population. So with the natives attached to the British Army and the regular European troops.

For each of them, as a class, there was a definite food-

supply per individual, based on the general stores of food in the country, and issued under the control of the military authorities.

Sometimes the burgher camps fared better than the troops; at other times the positions were reversed.

The evidence supporting the contention that scurvy is caused by a specific infection is derived from two sources—viz., study of the epidemic behaviour of the disease, and study of its clinical features.

The incidence of scurvy in the Eastern Transvaal will be considered in the following units:—

1. The burgher concentration camps.
2. The European soldiers.
3. African natives attached to the British Army.
4. African natives attached to the Civilian Repatriation Department.

With regard to class 1:

The *burgher camps* under special observation were those situated at Middleberg, Standerton, and Volkrust.

Each camp numbered about 5,000 inhabitants, of whom 48 per cent. were children under 12 years of age. The remainder were classed as adults, male and female, and were in the proportion of one to three. The inhabitants lived in tents, which were regularly arranged at wide intervals on sloping, well-drained soil, and each camp possessed its own internal medical and educational organisations. Every camp was surrounded by barbed-wire fencing, and was closely guarded; and as ingress and egress were privileges subject to military control, each camp was in a sense an open prison. No food could reach the camps, or be distributed to the inhabitants, except with official sanction. A definite scale of food, varying with the age of the individual, was laid down and rigorously adhered to. In each camp were separate quarters for the native camp inhabitants. These natives, who likewise had fixed rations, acted chiefly as servants to the members of the camp staffs and to the Dutch inhabitants. They averaged 150 per 5,000 whites.

The incidence of scurvy *at the burgher camps*, considered in detail, was as follows:—

1. In the camp at Standerton, between March, 1901, and January, 1903, there were no cases of scurvy.
2. In the camp at Volkrust, similar in every respect to that at Standerton, and over a similar period, there were no cases of scurvy.

3. In the camp at Middelberg, which was slightly larger than that at Standerton, there were 100 cases among Europeans, and 1 case among natives, over a similar period.

This outbreak of scurvy among adults at Middelberg, which occurred during December, 1901, and January, 1902, under a dietary similar in all respects to that in use at the camps at Standerton and Volkrust, immediately lends colour to the suggestion that the cause of scurvy is to be found in conditions other than those associated with food. The inhabitants of these camps had no occupation other than their domestic duties.

Secondly, with regard to European Soldiers.—The district under inspection as to the prevalence of scurvy among Europeans in British military employ was that of Standerton. All the sick within a radius of 40 miles were received into No. 17 General Hospital. This hospital had advanced up-country through Natal with General Buller's army, being known as No. 4 Stationary Hospital, and finally found a resting-place at Standerton as No. 17 General Hospital. It comprised 600 beds for Europeans, and a variable number—from 30 to 70—for natives in military employ.

Between December, 1899, and January, 1903, 22,000 European patients were admitted. This number included 100 sick Boer prisoners of war, 40 sailors of the Royal Navy, and 160 civilians in Government employment.

There was not a single case of scurvy among these admissions.

Between March, 1902, and February, 1903, three thousand soldiers around Standerton were examined while at their duties, and only one was found affected with scurvy. This was a private of the Scottish Rifles on detached duty, living with, and having charge of, the transport section of the native sanitary corps; and to his case further reference will be made subsequently.

Scurvy, therefore, with this single exception, was non-existent among European troops and Europeans in military employ in and around Standerton.

Thirdly, with regard to the Natives in Military Employ in and around Standerton, the evidence is derived from two sources: —

1. The statistics of the native Military Hospital attached to No. 17 General Hospital.

2. A repeated examination of the natives in military employ in the Standerton district.

Examination of the hospital record of admissions of natives shows that the first case of scurvy admitted occurred in November, 1901. This was the first out of a total of 300 admissions for all causes. Among these 300, however, were 18 cases admitted for indefinite symptoms: such as debility, rheumatism, inflamed connective tissue, and periostitis. These may possibly have been cases of scurvy. No further cases of scurvy were admitted between November, 1901, and April, 1902. In May, the last month of the war, 8 cases of scurvy were admitted: 6 from the Army Service Corps Transport Section on May 1st, and 2 from the Sick Horse and Remount Depôts a few days later. In addition there were 27 cases, diagnosable by their symptoms only, of the character above mentioned. The total admissions to hospital from all causes between November, 1901, and May 31st, 1902, was 390. Definite scurvy, therefore, formed 2 per cent. of the total admissions, and doubtful scurvy 7 per cent.

Between June 1st and September 30th, 1902, there were 193 admissions, including 22 cases of definite scurvy and 20 doubtful cases, *i.e.*, 11 per cent. and 10 per cent. respectively, of total admissions.

The last period noted is from October, 1902, to February, 1903. The total admissions numbered 86. Of these, scurvy accounted for 24, and there was 1 doubtful case. Definite scurvy, therefore, formed 28½ per cent. of all cases admitted during this period.

The following is a summary of the foregoing facts:—

Between January, 1901, and February, 1903, 970 natives were admitted to the Military Hospital. Before May, 1902, definite scurvy was practically absent. During this month, after a total admission of 600 patients, 8 definite cases of the disease occurred and were admitted to hospital. From this month onward, until February 7th, 1903, the prevalence of scurvy among the hospital admissions gradually increased from 2 per cent. to 28½ per cent. At the same time the total admissions for dysentery, diarrhœa, and enteric fever fell considerably, and the monthly admissions fell from an average of 57 to 21. It is to be remarked that this notable increase in the percentage of admissions of scurvy cases occurred on the cessation of hostilities, when the natives and troops to whom they were attached became stationary, and concentrated in fixed camps in and

around Standerton. It is of further importance to note that during the latter portion of the period under consideration, when scurvy was most prevalent, all restrictions in food had been withdrawn; and also all cases admitted came from units stationed in Standerton town.

Such is the information gleaned from the hospital statistics.

The increasing proportion of scurvy cases admitted to the native Military Hospital induced one to make a general inspection of all natives in military employ in and around Standerton. The men were examined in their own quarters while on duty. Outside Standerton, between March and July, 1902, before the troops and attached natives were concentrated in the towns, no signs of scurvy were found. After the concentration in Standerton, natives were again examined, and the following results obtained. Those inspected were of two classes, the labourers and the mule-drivers.

1. The labourers were divided into the Native Labour Corps and the Native Sanitary Corps. In the Labour Corps there were 117 men employed, and 16 per cent. were affected. In the Sanitary Corps 37 men were employed, and 76 per cent. were affected.
2. The mule-drivers were all attached nominally to regular regiments or corps.

The following is a brief statement of the incidence of scurvy among the mule-drivers:—

a. Attached to Pom-pom Section, 8 men. None affected.
b. Attached to Mounted Troops, 67 men. 22 per cent. affected.
c. Attached to Army Service Corps, Transport Section, 400 men. 32 per cent. affected.
d. Detachment in charge of Officers' Carts, Army Service Corps, 9 men. None affected.
e. Detachment employed as Police in Army Service Corps camp, 8 men. 4 affected.

To return to a consideration of the Native Labour and Native Sanitary Corps. These two units received exactly the same rations, which consisted of mealy meal 2¼ lbs. daily, and meat 1 lb. weekly. The Labour Corps had 16 per cent. affected; their work was that of heavy goods porterage at the railway station. On the other hand, the Sanitary Corps had 76 per cent. affected, and

their work was that of gathering together the carcases of animals that had died during the war around Standerton, and burying them in pits; their work was comparatively light. The eight men who were occupied in transporting the carcases by mule waggons to the burial pits were every one of them affected with scurvy, as was also the European soldier in charge of the transport party. This is the case referred to previously as the only European found affected with scurvy at Standerton. His food was the same as that of the other soldiers at Standerton, and was totally different from the food of the natives of the Sanitary Corps. Although he was granted the use of a separate tent, which stood side by side with those occupied by the Sanitary Corps natives, he, however, spent the whole of his leisure time with these natives, and associated intimately with them. The natives of this corps had been recruited but three months previously, direct from their homes, and had come in perfect health. It is a noteworthy fact that the head native of this gang occupied, with his family of six, a tent a short distance apart from the others, and although their diet was similar to that of the corps none of them were affected with scurvy. The greater cleanliness of this tent, as compared with the others, was also striking.

The marked contrast in the prevalence of scurvy in these two units, namely, 76 per cent. in the scavengers and 16 per cent. in the porters, under precisely the same dietary, again points to a factor other than food as the prime cause of the disease. Nor can it be entirely the unwholesomeness of the work of the Sanitary Corps, because other diseases due to insanitary conditions, such as diarrhœa, were markedly absent. It may be further mentioned that, although these two units, the scavengers and the porters, occupied separate quarters within a few hundred yards of one another, they were practically on one enclosed piece of land.

Next with regard to the mule-drivers. Their food was exactly the same as that of the ordinary European soldiers, with the exception of the lime-juice ration.

The Cavalry, Artillery and Pom-Pom Section natives lived in tents in the same camps as the European Cavalry and Artillery. Their surroundings were, therefore, comparatively cleanly, and the incidence of scurvy varied from total absence in the Pom-Pom Section to 22 per cent. in the Cavalry natives. On the other hand, the natives of the Army Service Corps, who lived in a large encampment

by themselves, and whose tents were uncleanly and compared unfavourably with those of the Cavalry and Artillery natives, had 32 per cent. affected. Of the 400 men examined belonging to this division, 200 were leaving for their homes on the following day, their contract period of service having expired. They complained of feeling too ill to work, and refused to sign afresh any contracts for further employment. Quite one-half of this number were suffering from scurvy, but the remainder showed no obvious signs of the disease. It will be said, however, that the regular troops were supplied with lime-juice, but not the natives. It may be pointed out, in return, that lime-juice was offered to all military natives in December, 1902, and the prevalence of scurvy was greatest in 1903. It must further be stated that the lime-juice was left severely alone by nine out of every ten men, troops and natives alike. This raw lime-juice was too acrid, too raw, and gave severe colic to many. It could not possibly be drunk without sugar, and sugar was too valuable for other purposes to be taken to improve extremely unpalatable lime-juice. The ultimate result was that the natives attached to these corps and living with them, had precisely the same dieting as the English troops. Still only the natives were found affected by scurvy, and that corps of natives (the Army Service Corps) was most affected, whose camp was most unclean and least under direct military supervision.

Here, again, proof seems to be wanting that the food factor is the cause of scurvy. In this connection, further, the condition of affairs in the 19th Hussars is interesting. One squadron of the 670 Europeans forming the regiment were buying fresh vegetables regularly, in addition to their ordinary rations. The remaining two squadrons subsisted on the rations issued: there was no sign of scurvy in either Section.

Fourthly, the last unit in which inquiry was made as to the incidence of scurvy, comprised 900 natives of Africa employed as drivers and leaders of mules by the *Repatriation Department*—a civilian organisation inaugurated on the cessation of hostilities. The sick of this corps were treated at the hospital in the Burgher Camp, Standerton, when too ill to look after themselves; but, when merely unable to work, were kept in sick detention tents close by their regular quarters.

Between August, 1902, and February, 1903, the Department being in full working order, 80 cases of all diseases

were admitted to the hospital. Of these 50, or 62 per cent., were cases of scurvy. In February, 1903, a final inspection of the natives of this Department was made. Of the 900 employed 500 were examined, and 252 were found to be affected, *i.e.*, 50 per cent.

Of the 14 cases in hospital 12 were scurvy; of the 14 in the sick detention tents 12 were scurvy. In connection with this inspection, the diagnosis of the cases was confirmed by the senior medical officer of the Burgher Camp, Standerton (Dr. Alexander).

Again, the feature to be noted is, that the food of these natives was quite as wholesome and plentiful (in some respects more so) as that supplied to the natives under military control; but, on the other hand, their camp was the least tidy and clean of all at Standerton. The tents were overcrowded, closely packed together, and arranged with a total disregard for sanitation. The area of this camp, containing 900 men, was only one-third in extent of that occupied by the Scottish Rifles, who numbered 500. The statistics of the sick in detention tents were not kept, or the tale would have appeared heavier. Although the natives of the Repatriation Department did not outnumber by any means the natives in military employ, the incidence of scurvy amongst them was the heaviest of all native units at Standerton. A consideration of this fact, then, that this body of natives with the best and most liberal food supply in the town had the heaviest incidence of scurvy, again points to the conclusion that food as a factor in the production of the disease must be eliminated.

Further evidence against food being the prime cause of the condition is afforded by clinical study of the cases—more especially by consideration of the mode of onset and the results of treatment. All these cases of scurvy commenced as an inflammation of the gums, and the general symptoms followed at varying intervals, but always secondarily to the gum condition.

Improvement or the reverse followed *pari passu* with improvement or aggravation of the gum condition. This inflammation of the gums occurred in all cases, without exception, and in a definite manner. The process was as follows: Around the tooth and next to the gum was first noticed a collection of food. The gum-edge nearest this aggregation soon became of a deeper red tint. This deeper tinted area spread rapidly, but in the early stage was

always separated from the healthy gum by a definite violet-mauve demarcation line. In a few days the whole of the gums, both internally and externally, became involved in the inflammatory process, and areas devoid of teeth which had hitherto been healthy were now affected At this stage the affected tissues were deep-red in colour, soft, swollen, and compressible, tender, and bled easily on pressure. In a day or so the gums became easily separable from the teeth; and deep down, between the gums and the teeth, definite masses of a cream-cheesy consistency and colour could be seen and removed. These masses, on microscopic examination, were found to consist of pus cells, numerous micro-organisms of various kinds, and food *débris*. At this stage there were no constitutional symptoms, nor could any changes be detected in the cardio-vascular system. These conditions developed later at varying intervals, and it was found that they did not develop when means were adopted to improve the gum condition. Moreover, in cases in which the general disturbance was present, there could be no ultimate recovery without a prior return of the gum condition to the normal. The method of treatment adopted, therefore, consisted of rest in the open air, isolation from previous surroundings, coupled with rigorous and frequent mouth antisepsis, care being taken to remove the cheesy masses mentioned above. The rate of general improvement and return of the gums to the normal condition was in direct proportion to the amount and efficiency of the antiseptic treatment employed for the mouth.

The fact that these cases when seen early could be completely cured by this treatment alone—with, be it noted, no alteration of any sort in the diet—argues directly against the diet being the prime cause of the condition. Further, the value of local treatment for the mouth condition is emphasised by a comparison of the results of treatment in advanced cases by (1) local plus constitutional treatment; and (2) by constitutional treatment alone. For local plus constitutional treatment, the average length of residence in hospital was from twenty to thirty days; but, from constitutional treatment alone, from sixty to ninety days. It is also interesting to note that in advanced cases in which mouth antisepsis was employed there was little or no pyrexia, but that periods of pyrexia were a feature of similar cases in which this treatment was omitted.

These clinical facts, therefore, seem to suggest that the condition is not due to the food, but is one caused by an

infection through the gums and mouth. Whether the system is infected directly through the gums or through the swallowed saliva after the gum condition has developed, cannot be readily determined.

Further, that the condition was found to prevail only among natives concentrated at Standerton, and then only in direct proportion to the absence of discipline and European habits of cleanliness, points to the fact that the unchecked habits of natives must in some measure either account for, or at least increase, the liability to the disease.

The natives seldom used soap, and rarely did they wash their hands or faces; and a large proportion, though they wore little or no clothing, were extremely verminous. This clothing was second-hand, and usually consisted of one or more cast-off European garments, which were very filthy and seldom washed. They rarely washed their blankets, and never swept out their tents. They had common eating and drinking utensils, and frequently several ate out of one dish at the same time. Though dental caries is less common among them than among Europeans, they rarely washed out their mouths. The majority of the natives chewed tobacco, "black cavendish" being easily obtainable through the troops; and not infrequently they passed the half-chewed "quids" from one to the other for further use. With this chewing there was expectoration free and plentiful—a habit found impossible to repress. Then they used mouth-organs and similar instruments in profusion, passing them from one to the other. They left both half-eaten food and food in preparation lying about on the ground, both inside and outside their tents. Moreover, the tents were always in use, and crowded during their off-duty time. Altogether, these habits formed an excellent basis for the conveyance of any mouth-to-mouth infection.

To sum up—

1. The outbreak at the burgher camp, Middelberg, among Europeans and natives, was attributed to the nature of the food supplied. It was noticeable that the European sufferers consisted mainly of those persons who from advancing years were unable to fend for themselves, and of those who, suffering from mental depression at the prolonged separation from relatives, neglected to obey even the ordinary rules of personal cleanliness. Moreover, the cause could not have been in the food, seeing that food of precisely similar nature was in use at Standerton and Volkrust, and yet scurvy was entirely absent from these latter camps. But, on the other

hand, the sanitary condition of the Middelberg camp was much inferior to that of the other camps named. At this time a determined effort was made to improve the sanitary condition of camps. Soap and fuel were ordered to be issued in unlimited quantities, whereas previously they had been supplied in restricted amounts. These articles were included in a new ration-scale, which was also introduced at this time. Scurvy now disappeared from Middelberg. It may be said that the cessation of the disease was due to the new diet provided; but the total absence of scurvy under the old dietary scale at Standerton and Volkrust is entirely opposed to this view; and it is remarkable that the disappearance of the disease was as markedly coincident with improved personal cleanliness in the Middelberg camps as with the change in diet.

II. Scurvy did not appear in the district of Standerton until the last month of the war, when the mobile troops became stationary and concentrated into large units. Further, it continued with increasing severity among these units when the war had ceased, and when restrictions in the way of purchasing extra food had been withdrawn. After the war, all classes were free to supplement their Government rations by purchasing fruit and vegetables at the various provision stores in the town. Special inquiry was made among those showing well-marked signs of the disease, as to whether or no fresh fruit or fresh vegetables had formed part of their dietary in addition to the official supply of rations. In many cases there was abundant satisfactory evidence forthcoming to the effect that the sufferers were purchasing and consuming fresh pine-apples, fresh bananas, or fresh melons and other vegetables imported direct from Natal. It is highly probable that these Natives, whose daily pay always equalled, and in many cases exceeded that of the English soldier, both purchased and consumed regularly a larger quantity of fresh vegetables or fruit than was the custom of our British European troops.

III. The only European affected at Standerton was attached to the Native Sanitary Corps—a unit of which 76 per cent. suffered from scurvy. Let it be noted, however, that this European lived with these natives. His diet was similar to that of the other European soldiers at Standerton, but he spent the whole of his time in the company of these scavengers; and, by reason of this mode of life, was freely exposed to any mouth-to-mouth infection. On the other hand, the family of the head native, consisting of six

individuals, was unaffected by scurvy. Their dietary was that of the Sanitary Corps, but they lived in a tent at a short distance from the scavengers' encampment, and their standard of personal cleanliness was of a high order.

The varying incidence in the two units—the Native Labour Corps and the Native Sanitary Corps—namely, 16 per cent. in the former and 76 per cent. in the latter, cannot be explained by the food alone, since both units were on precisely the same diet.

IV. With regard to the other natives inspected at Standerton, not one of the 150 employed at the burgher camp as servants to the European camp staff was found affected; nor did natives employed as domestic servants in the European Military Messes, shops, or households in the town, show signs of the disease; but in the remaining units, both civil and military, scurvy prevailed directly as the filthy habits of the natives were allowed to go unchecked by direct European control.

V. Of those cases of scurvy admitted to the Military Hospital at Standerton, 12 per cent. died. Of those who recovered, 90 per cent. went back to their former duties, their former dietaries, and their former surroundings; and though they lived among men afflicted with scurvy, yet the hospital records do not show a single instance of a case recovered from scurvy seeking readmission for a recurrence of the disease.

These facts, therefore, concerning the incidence of scurvy in the various bodies of individuals seem to be against the contention that food is the sole cause of the disease; and, moreover, the clinical facts also would seem to oppose the contention.

The infection appears to occur through the mouth and gums, the general system being involved later and secondarily. The manner in which the primary infection occurs, and how it gives rise to the manifestations of the disease in the mouth, are points on which it is not easy to make any definite statement, and require further investigation.

In conclusion, Gentlemen, I submit that the facts adduced seem to prove that scurvy is not due to either the presence or absence of any particular kind of food, but rather to an infection for which food may act as a vehicle under conditions of dirty storage or dirty preparation; and, considering that the disease prevailed in inverse proportion to the

standard of personal hygiene of the individuals affected, its infectivity would seem to depend on the insanitary habits, the degree of intimacy, the overcrowding, and, perhaps, the unwholesome occupation of those who were its victim.

WEEKLY DIETARY SCALES AND INCIDENCE OF SCURVY.

A.—*In use at all Burgher Camps till December 31st, 1901.*

Adults ... —Flour, 7 lb.; sugar, 12 oz.; ground coffee, 6 oz.; salt, 4 oz.; tinned meat, 2 lb.

Children (6-12).—Flour, 7 lb.; sugar, 12 oz.; ground coffee, 3 oz.; salt, 2 oz.; tinned meat, 1 lb.

Children (1-6). —Flour, 3½ lb. Remainder, as children (6-12).

Note: When fresh meat served out—Adults, 3 lbs.; children, 2 lbs. Tinned milk issued, "made up" by dispensers with water, ready for drinking, for children only, and issued daily; but for adults only on Medical Officer's order. Wood and coal issued when required.

B.—*Introduced to all Burgher Camps, January 1st, 1902.*

Age.	Vegetables.	Flour.	Syrup or Sugar.	Coffee.	Fresh Meat.	Salt.	Milk.*	Rice.	Soap.	Butter.
	lb.	lb.	oz.	oz.	lb.	oz.		lb.	lb.	oz.
Adults... ...	1½	7	12	7	4	4	1 tin	1	½	4
Children, 6-12 .	1½	5	12	4	3	4	7 quarts*	½	½	4
Children, 2-5 ...	As supplied by Camp Matron	3½	12	—	2	2	14 „ *	½	½	4
Children under 2 years ...		2½	12	—	—	2	14 „ *	—	½	4

* Tinned milk, made up by dispensers with water, daily ready for consumption.

A + B.—*Incidence of Scurvy.*

Camp Populations—Children, 48 per cent.; Adults—Male, 12 per cent.; Female, 40 per cent.

Burgher Camp, Volkrust.	Population,	5,000.	Nil.
„ „ Standerton.	„	5,000.	Nil.
„ „ Middelberg.	„	6,000.	Europeans, 100 cases.

Outbreak in December, 1901, and January, 1902.

C.—*European Soldiers—at Various Times.*

Bacon or cheese, jam, frozen meat, fresh meat, tinned vegetables and meat, coffee, sugar, salt, rum, soap and fuel, tea, lime-juice (not usually taken).

Admissions to No. 17, General Hospital, Standerton, 22,000. *Scurvy, nil.*
Examined on duty, 3,000. *Scurvy, one.**

* This man was in charge of transport section of Native Sanitary Corps, F.I. 2.

D.—*For Natives Employed in Burgher Camps: Standerton, Middelberg, and Volkrust.*

Flour, 7 lb. ; sugar, 8 oz. ; coffee, 4 oz. ; salt, 4 oz. ; meat, 1 lb.

Total employed at each camp, 150.

Scurvy at Middelberg, *one case.*

„ at Standerton and Volkrust, *nil.*

E.—*For Natives Employed by Military as Muleteers.*

Lime-juice issued in December, 1902, but not taken.

Bread, flour, or biscuits, 7 lb ; sugar, 14 oz. ; coffee, 3 oz. ; meat, 7 lb. ; salt, 2 oz. ; tinned vegetables bi-weekly, as for European troops.

Attached—1st Essex, Rifle Brigade, Leicesters. 60 examined (July, 1902) outside Standerton. *Scurvy, nil.*

Attached—Army Service Corps Transport *at Standerton*, 1903.
Officers' Cape Cart line, 9 men. *Scurvy, nil.*
Police ... ... 8 men. *Scurvy*, 4.
Muleteers, 400 examined. *Scurvy*, 32 per cent.

Attached—Pompom Section, 9 men. *Scurvy, nil.*

Attached—19th Hussars and 83rd Field Battery R.A., 63 men. *Scurvy*, 22 per cent., *at Standerton*, 1903.

F.—*For Labourers Employed by Military as Porters and Scavengers.*

Mealy meal, 16 lb. ; meat, fresh, 1 lb. ; lime juice, 1 oz. weekly in December, 1902, but not taken.

Excess* mealy meal, fermented = a drink called "Mahow" (intoxicating).

* Can be burnt and drunk as a black liquor = native coffee.

I.—*Native Sanitary Corps = Scavengers. Light Work.*

February, 1903, at Standerton.

1. Examined, 34. *Scurvy*, 76 per cent.
2. Transport,* 7. *Scurvy*, 100 per cent.
3. Later examined, 20. *Scurvy*, 100 per cent.
4. Head native with family of six, living separate. *Scurvy, nil.*

* To the Transport section is attached European under Dietary C, who was affected with scurvy.

II.—*Native Labour Corps = Porters. Heavy Work.*

Employed, 120. *Scurvy*, 17 per cent. Examined, February, 1903, at Standerton.

G.—*Natives Employed by Repatriation Department, Standerton, as Muleteers.*

Biscuits, flour, or meal, 7 lb. ; meat, chiefly fresh, 7 lb. ; coffee, 7 oz. ; salt, 3½ oz. ; lime juice, 1 oz. ; sugar, 21 oz. ; wood, 14 lb.

Employed, 900 ; examined, 500 ; scurvy, 50 per cent.
In hospital, 14 ; scurvy, $\frac{12}{14}$ = 86 per cent.
In detention tents, 14 ; scurvy, $\frac{12}{14}$ = 86 per cent.

NOTE.—All food supplied during 1902 and 1903 of uniformly excellent quality.

DISCUSSION ON DR. COPLANS' PAPER.

THE PRESIDENT: I am sure we have all listened with very great pleasure to Dr. Coplans; he has given us a very instructive and interesting view of scurvy, and I shall be glad to hear any discussion upon this very original Paper. I am sure there are many points in it which are well worthy of it. Perhaps Professor Wright will open the discussion.

PROFESSOR WRIGHT: I quite agree with what you have stated about the lecture to which we have listened. It was marked by originality of thought. Dr. Coplans has certainly thought the matter out for himself. I am not disposed, however, to think that he has been right to think out the whole question for himself. I do not think it is justifiable to take up the position that Dr. Coplans has done, and to say: "I am cutting myself off from all previous experience; I shall examine this matter *de novo*; I shall base my conclusions only upon what came before me in South Africa."

In the first place, I must point out that the association of scurvy with dirt cannot be sustained. All natives are charged with being dirty; yet there are many regions in which you have dirty natives, and an absence of scurvy. On the other hand, we find scurvy in England attacking children of the rich, reared in the cleanest and most sanitary nurseries. I suggest that Dr. Coplans is led into error by saying: "I will not look beyond the particular circumstances in which I found scurvy to occur in South Africa."

Again, we have not had all the facts with regard to the dietaries in the camps placed before us. Dr. Coplans tells us that all these people were subsisting on the same diets, and that none the less scurvy broke out in certain camps, while it failed to put in an appearance in others. But to supply people with a certain dietary is not always to ensure that the dietary is eaten; one cannot be quite sure that in every case the food which was furnished was actually eaten. I have lately seen a lady lapse into an aggravated condition of scurvy in a nursing home, where, no doubt, supplies of

anti-scorbutic food were delivered daily by the greengrocer. Again, though nominally the same, the food may have been of different qualities in different camps. There may, for instance, have been fresh meat and milk in one camp, and preserved meat and milk in another; or, where two camps had preserved food, the brands of food issued may have been different in the two cases. Therefore, I do not think we can be sure that the camps which Dr. Coplans speaks of had all the same dietary.

I can quite believe there are other circumstances than food that may make people more inclined to fall victims to scurvy. The question of muscular exertion may, for instance, have an importance. In the voyage of the *Centurion*, we read that the exertion of working the ship, even the exertion associated with their being moved on to shore, aggravated the scorbutic condition, and was followed by fatal results.

Dr. Coplans has suggested that scurvy is a bacterial disease. All diseases at their close become bacterial diseases. I have had the opportunity of seeing cases of scurvy at Netley in its most aggravated form. The patients in question had been invalided home after the relief of Ladysmith. I have no words sufficiently forcible to depict their clinical condition. Their appearance was that of corpses which had been buried and dug up again. Their eyes had sunk back into their sockets, their whole bodies were covered with purple *ecchymosis*. One of these patients was the victim of an enormous bed-sore. He had, in addition, an abscess of the size of an orange sticking out, after the fashion of a cheek-pouch, from his jaw. While these symptoms testified to a bacterial infection—and, in the case I refer to, to a fatal bacterial infection—that infection was only consecutive to a blood alteration.

The idea of making antiseptic precautions, as Dr. Coplans has suggested, the basis of a treatment of scurvy, seems to me dreadful. I think that while such precautions might for a time keep a bacterial infection at bay, the patient would none the less die for the want of anti-scorbutics.

When I speak of a blood change in scurvy, I have in my mind a very definite picture of that blood change, and nothing in pathology is to my mind more certainly established, than that the essential essence of scurvy is to be found in a diminution of the alkalinity of the blood. Since I have been at St. Mary's, I have, through the kindness of my colleagues, had opportunities of making observations on the blood of some eight cases of infantile scurvy. In each of

them, as in the cases of scurvy already referred to which came under my notice at Netley, I found a decrease of the alkalinity of the blood. If no question can arise as to this, it is, I take it, absolutely clear that it is, as was originally suggested by me in a Paper which appeared in the Army Medical Report for the year 1895, and in the *Lancet*, August 25th, 1900, an acid-intoxication. Now, if that is so, then the scorbutic condition must be present in many cases where the clinical symptoms are not yet manifest. I have seen such cases in infants: cases where the child was out of health, but where the periosteal hæmorrhages, the swelling, and the characteristic symptoms, had not yet appeared, though the conditioning cause was already present. When I consider these facts, and then hear from Dr. Coplans that one camp showed many cases of scurvy and another camp none, it occurs to me as a possibility that there may merely have been a difference in degree as between the camp which suffered from overt scurvy and the camp that is said to have been free from scurvy. My friend, Dr. Dodgson, who examined the blood of a great number of natives in South Africa during the war, in camps which furnished only a few overt cases of scurvy, found among those who showed no characteristic symptoms a number of cases in which the alkalinity of the blood was diminished.

I come to the next point. In the Paper in the Army Medical Reports above referred to, I considered the composition of the ash of the principal food-stuffs, and I showed that the ash of scorbutic food-stuffs contained mineral acids in excess; and I pointed out that a dietary consisting exclusively of these acid food-stuffs would impoverish the blood in alkaline salts. It is, however, a question of time.

We can all subsist upon a scorbutic dietary: upon a dietary of salt meat, pork, and cereals, or cereals alone, for a certain time—for a certain number of weeks or months; but if that dietary is not changed after a certain time, we begin to suffer from scurvy. In the same way, it takes a period of some weeks upon a dietary of cereals to effect the introduction into the system of an infant of a quantity of acid such as will be capable of reducing the alkalinity in the blood to a serious extent. One works up slowly to that. We may bring into connection with this Dr. Coplans' observation that during the first year there was no case of scurvy in the camps upon the official dietary. Afterwards, he tells us though the dietary was continued, scurvy broke out. The dietary is not exculpated by the fact that it did not immediately produce scurvy.

With regard to the treatment of scurvy, I may point out that the anti-scorbutic treatment which is resorted to, *i.e.*, the administration of potatoes, and similar vegetables and fruits, and raw meat juice, is scientifically justified as supplying all the elements which are turned into alkalies in the body. I have, however, urged the substitution of the essential anti-scorbutic elements for the raw materials ordinarily administered; and I have verified that the defect of alkalies can be restored to the blood in this manner within as many hours as it would take weeks on the system now in force. I have employed in particular lactate of soda, making choice of this organic acid salt because of all acids, lactic acid is the acid most easily converted into carbonic acid in the body. Lime juice ought to be eschewed as containing citric acid. I take it that this citric acid, inasmuch as it breaks up into CO_2 and H_2O without leaving any residue, must be entirely useless as an anti-scorbutic. Citric acid can only decalcify the blood. I think, inasmuch as lime juice contains, in addition to the citric acid, only a nominal quantity of citrates, the soldiers referred to by Dr. Coplans were well advised to avoid taking their lime juice.

MAJOR FIRTH, R.A.M.C.: Following the very erudite and comprehensive statement of Dr. Wright, I feel that I am put to some disadvantage. As to the Paper which we have heard, I personally cannot accept Dr. Coplans' conclusions in their entirety, because I think they are somewhat incomplete; but we are distinctly indebted to him for the Paper, inasmuch as he has put before us the possibility of working on original lines, and of taking an original—if somewhat unorthodox—view of this question of the pathology of scurvy. While I think very few of us are prepared to question the accuracy of his statements, it is legitimate to question the deductions he draws from them. For my own part, I think the pathology of scurvy is adequately explained, though its etiology still presents some obscurities.

The first thing in this Paper that struck me was, that we have no guarantee or any information concerning the nature of the foods issued, as regards their general quality and freshness. I took the trouble to work out some of these dietaries which are given in the Appendix, and was surprised to find the extraordinarily low value they have when put into ordinary dietetic terms of so many calories: the average dietary that these men were getting, roughly

speaking, was only worth 2,000 calories. We know what the internal needs of the body at rest require that men should have, and we know that these Kaffirs were called upon to perform heavy labour: therefore, we have primary evidence that these men were inadequately fed. But even admitting that they were getting a sufficiently high dietary, suitable to meet the needs of their bodies, what guarantee have we that the materials constituting their daily ration were fresh, or that they were of the same nutritive value in the various communities? Look down these dietaries, and you will find that the consumers depended largely on cereals, in the case of those employed as porters and scavengers, and that they only received 1 lb. of meat in a week. If you come to look at the nature of these dietaries in the degree of their component parts, the nutritive value is absolutely inadequate. Again, in the case of those who received milk, what guarantee have we that these samples of condensed or preserved milk were anti-scorbutic? A large number of samples of preserved milks have, from time to time, come under my examination; and I entertain no doubt that, in the case of many of those now in the market, the nature of their ash has materially altered from that of fresh milk. Instead of having ashes which are clearly alkaline, it is not unusual to find in certain old and inferior brands that the ash is distinctly acid. I admit that there may be nothing much in that, but it is a small straw which may give us a hint as to which way the wind is blowing. If that be so, if there was an issue of any of these degraded milks—and it is not unreasonable to think that the authorities did not give to these natives the best brands of condensed milks—it is not difficult to trace the possible origin of the scorbutus. But quite apart from the milk issue, I am inclined to think the ration as a whole was absolutely inadequate to act as an anti-scorbutic. We know that this question of condensed milk and treated milks is open to very grave suspicion amongst clinicians in civil practice; and that it is a difficult matter, even in a civilised community, under stable conditions, to avoid the scorbutic taint among children; therefore, how much greater a difficulty does this question present in unstable conditions, as in warfare, and where you are dealing with native races. We do not know much yet about the changes which take place in foods during the process of preservation; and considering how large a part preserved foods are now playing in the feeding of people, it appears to me that we should pause before we dogmatise as

to the etiology of scurvy. If we have reasons to suspect grave molecular changes in a single article of preserved food, such as preserved milk, may it not be the same in respect of tinned meats, dried mealies, and the various cereals of unknown age and history? In fact, the whole question is so complex that we may say we are only on the threshold of knowing the actual facts concerning the true dietetic value of preserved food-stuffs.

Notwithstanding the dexterity with which the evidence has been handled, I confess to being not altogether satisfied with the plea put forth. As regards the pathology of the disease, we are bound, I think, to accept what Dr. Wright has told us as being sufficiently clear and obvious; but as to the etiology of the affection, I must adhere to the view that, in the light of our present-day knowledge, the actual foundation of scurvy, whether in natives or whites, is the continuous use and consumption of degraded foods: that is, foods which have lost the essential characteristics of the fresh articles. It is quite clear that much work has still to be done concerning the etiology of scurvy. Only this week, I have had a letter from a well-known member of this Society—Dr. Parkes, of Johannesburg—in which he mentions the great prevalence of scurvy among the native races of South Africa. I feel sure that, in the reading of this Paper, Dr. Coplans has done well by directing attention to the vast field for research which still needs to be exploited in the direction of practical dietetics.

PROFESSOR KENWOOD: Mr. Chairman and Gentlemen, I did not intend, to say anything this evening. Scurvy is not a subject with which I have any personal acquaintance, and I had come to listen and to learn. I have read Dr. Coplans' Paper, and listened to it this evening; and I am bound to say that he does seem to me to have produced a very great deal of evidence which certainly is very much opposed to the purely food theory—the theory of the absence of fresh foods to which one so generally finds scurvy attributed.

Those are very interesting facts to which Professor Wright has called attention; but there still remain several facts in this Paper which have not been answered by any speaker yet.

First of all, although it is perfectly true that Dr. Coplans has not been able to analyse all his food-stuffs, and that some may have been of bad quality, I do not look upon that as seriously prejudicing his conclusions, because he

deals with communities which have been under his actual observation for long periods, and communities which have been placed in the exceptional circumstance that he has been able to keep a close tally of all the food available; and if the food has varied in quality, it obviously should affect equally all sections of the community who have partaken of that food; yet he says that in one section placed in insanitary conditions, and which, I think, he is prepared to maintain was fed on the same rations as another section who did not perform such insanitary functions, the incidence of scurvy varied from 76 per cent. in the former to 17 per cent. in the latter. That argument has, I think, not been met by any speaker up to now.

Again, Dr. Coplans' statement that scurvy appeared amongst the body of natives who had the best and most liberal supply of food seems to me to be a matter which it is difficult to account for on any purely food theory. Personally, I should feel disposed to attach great importance to the very exceptional experience which Dr. Coplans has had, and to the very great evidence which he brings forward in support of the contention that there is something more than the food question involved in the etiology of scurvy. So far as I am concerned, he has convinced me on that point.

As to infantile scurvy in this country, I think there is some scope for difference of opinion as to whether the infantile scurvy which we see in this country is precisely the same disease of which Dr. Coplans is speaking; and as to the successful treatment of scurvy being entirely restricted to anti-scorbutic drugs. Here again we have Dr. Coplans' experience that he only applied antiseptic local treatment, and that the patients got well.

It is well to be a little diffident in dealing with such matters as the causes of disease, when one considers the change of front that we have had to make on so many matters during the last few years. I think, if I had got up in this room twenty years ago, and said that influenza was not largely air-borne, you would probably have said that I ought not to be in this particular building, but in some other where I should be under suitable control; or, if I had said that malaria was disseminated by the bite of a mosquito. I think it is not improbable that the purely food theory of scurvy causation may possibly be relegated to the same place as the theory of the aerial convection for long distances of small-pox will be in, say, ten years time.

I thank Dr. Coplans for a very interesting and suggestive paper.

COLONEL LANE NOTTER, R.A.M.C.: I do not know that I have much to add to what has been said by the previous speakers. Unfortunately, the statistics given relate to events which have occurred after I left South Africa, and therefore I am not personally acquainted with them. I know the camps there which Dr. Coplans has mentioned, and there are one or two points in connection with his Paper on which I think I might enlarge.

First of all, as to the food of the native in South Africa. Now, it is true that the native received a Government ration, but he is one of the foulest of eaters. Any decomposed food, and especially the entrails of animals, form his choicest meal. It is possible he gets into a condition of blood-poisoning, and this condition of the gums is then only a secondary affection. I think such is extremely likely. I know with regard to the camp at Middleberg, because I had something to do with that at one time: the people there on some occasions drew tins of jam per unit in excess for every person in the camp; so that, so far as anti-scorbutics were concerned, they had plenty in the camp at Middleberg, but I do not know whether the natives were as fortunate as the burghers in their camp had been.

Another point in connection with the theory which Dr. Coplans has put forward is this: I take it that if it were a bacterial infection, it would be present also in the white population. There would not be one section entirely free: more white men would be attacked; but, as it was, the scorbutic element was never present to any great extent. No soldier suffered from it, for in no campaigns have the supplies been better than they were all through the South African campaigns.

Dr. Coplans also mentioned the fact that the natives who were employed as officers' servants and in messes—that these men were not affected at all. We can understand that: we have all suffered from their pilfering habits, and no doubt they took care of themselves at the expense of their masters.

Standerton was an extremely unhealthy place. I think, without exception, the water supply at Standerton was about as bad as it could be. The intake was from the river, where, to make the pumping more efficient, the length of pipe up the river was cut short; so that actually within a few yards of a drain the water was pumped up to

the reservoir at the top of the hill. The whole of the camping-ground there was more or less affected, and was not in a cleanly or good state.

I still think that, although there is much of interest in this Paper, the symptoms of scurvy were only a secondary affection, and that a good deal that the native suffered from was from anæmia: the result, no doubt, of insufficient and bad food to a large extent: the conditions Dr. Coplans describes were more or less connected with this, and can hardly be taken as instances of true scurvy, although bearing a strong resemblance to it.

LT.-COLONEL DAVIES, R.A.M.C.: I have very little to add to what has been said this evening on this matter.

I should only like to give a few facts gleaned from certain statistics. Of course, it must always be remembered that the diagnosis may have been faulty in previous days. As a matter of fact, we have not yet the medical statistics with regard to the Army in the late war. I have made the best inquiry I can, and I am told that, practically, there was no scurvy amongst the British troops.

We have had very little opportunity of seeing scurvy in the Army of late years, either on board ship or anywhere else, except in some parts of India, where there have been small and limited outbreaks. I can recall three small outbreaks at different times. The only explanation that could be given in two instances* was, that they were drafts of troops who had arrived from home, and had just come off the ship. On board ship there had been the usual deficiency of vegetables, and that was put down as the cause of their scurvy; and in each instance when fresh vegetables were given to them, the scurvy immediately disappeared. There was one outbreak† where there was no deficiency of vegetables, or of food generally. They had potatoes and onions every day, and plenty of good fresh meat; and yet there were 56 cases of scurvy, all occurring during the rains, when people are more inclined to take any microbial infection, if there be any about. Directly the rains came to an end, and they were able to get out and exercise themselves, they recovered marvellously and immediately. In a few other cases where scurvy has occurred in India, particularly at Quetta on two or three occasions, it was put down to the difficulty of getting

* At Rurki, in 1885, 32 cases; at Poona, in 1886, 15 cases.

† At Chakrata, in 1886, 56 cases.

vegetables, where vegetables were not grown. Throughout India, one does not get fresh vegetables as a rule; but in Quetta they are very scarce, and three times scurvy has broken out there owing to that cause, it is said.

In the first Boer War vegetables were very difficult to get; and there was, in consequence, a small outbreak of scurvy at Ladysmith (1881), when 25 cases occurred.

It is curious to observe that scurvy has never occurred in the Madras Presidency; you do not get very much vegetable food in the Punjab, but in Madras everybody has plenty of vegetable food, and it is noteworthy that scurvy has always been practically non-existent there.

There is one other old fact to which I should like to allude. Sir Thomas Longmore, in his pamphlet: *The Sanitary Contrasts of the British and French Armies during the Crimean War*, drew an instructive comparison between the health of English and that of French troops, in the first and second winters of that campaign. In the first winter, 1854-55, the British suffered severely. There were 291 deaths from scurvy and scorbutic dysentery, the troops being 31,000 strong. Amongst the French, who numbered 79,000, there were only 145 deaths. Of course, the sanitary conditions of the camps were very different also. But what was the cause of the much greater amount of this illness amongst the English than amongst the French? It was put down to the fact that the French soldier was better able to fend for himself, and to cook for himself; and in particular (probably from experience in Algeria) knew how to make use of any kind of wild plant he could get hold of; any kind of weed he could pick up, he would make a salad of, and eat it with his food; whilst the British soldiers were living entirely on salt meat and biscuit, without any vegetables at all, for a considerable time.

Then, on coming to the next winter, we find that whereas we had had 291 deaths in the first winter, in the second there was only one death from scurvy amongst the British troops, although their numbers had increased to 50,000. What was the explanation of that? The hygienic conditions were improved generally; but in particular with regard to food supplies, everything was supplied that could be supplied; as I suppose was the case in the late war: absolutely everything that money could purchase was provided. But with regard to the French, they retrograded—they went back. Their strength was then 131,000, and they had during the second winter 964 deaths from

scurvy. Why they should have lost their fending and cooking capacities during this later period, I am unable to say. It was certainly the case that their hygienic conditions were not so good in the second winter as in the first. The troops were also much enfeebled and overworked; and it is said their rations were coarse, not varied, often insufficient, and deficient in vegetables. They were still under canvas, while our men were housed in wooden huts.

DR. WILLOUGHBY: I must confess to great disappointment with this Paper: for with such opportunities of observation, where the food was supplied by Government to natives and Europeans alike, there seemed to be all the conditions requisite for exact experiment on a gigantic scale. But the very vastness of the field seems to have militated against his deducing positive results.

Of course, Dr. Coplans can give no guarantee as to the quality of all the food supplied.

There is one thing I would like to ask: Has no one heard of Professor Tonk, of Christiania, and his theory of the causation of scurvy? We have had plenty of destructive criticism, but we want something constructive, and based on practical observation. We have in the observations of the last half-dozen Arctic expeditions the most conclusive evidence, positive and negative, against the theory that any certain article of food is causative of scurvy, or that any certain article of food is a preventative. I do not dispute the theory of acid intoxication as a pathological factor, and perhaps a proximate cause; but the necessity of vegetables as an article of diet has been exploded. We have simply to take the stories of the last half-dozen recent Polar expeditions to see this. Professor Tonk looks upon the pathology of scurvy as simply a chronic ptomaine poisoning; and that seems to me to explain all the circumstances.

There are many cases in which salt pork and tinned meats have formed the main supply of food, and yet no scurvy has occurred; and when we come to cases in particular, we find Nordenskjold's and Nansen's expeditions on the high seas, all living entirely on preserved foods, and escaping; whether or not preservation destroys the anti-scorbutic properties, there was no decomposition in Nansen's case, owing to the great care which he took. We have the case of Leigh Smith's expedition in 1881 to Franz Josef's Land, where twenty-eight men spent a whole year under the most miserable

conditions, living entirely on bear's flesh; yet they had not a trace of scurvy amongst them. We have Nansen and his companion for a whole year living in the same way. We have the still more interesting observation of the Jackson-Harmsworth expedition, where the exploring party, living on the produce of their guns, subsisting on fresh and even raw bear's flesh, with no vegetable food whatever, and escaping entirely, while every one of those who remained on board ship, though they had lime juice every day, suffered more or less from scurvy—two died; but it was noticed at the same time that the meat, when the casks were opened, was always more or less high: sometimes the men refused it, but sometimes they ate it, although they would not take the bears' flesh.

Then we have the case of the Lapps in Finland, who have plenty of vegetable food, but who from choice eat their fish always more or less putrid, and they suffer severely from scurvy.

As regards infantile scurvy, I had one extreme case in my practice, some years ago. The parents were persons in easy circumstances, most careful and attentive to their children, all of whom had good health but this one: its diet having been exclusively revalenta and condensed milk. The other children had been brought up by hand in various ways, and were all healthy.

Whatever be the relation between adult and infantile scurvy, the comparatively few cases we have of the latter are among children brought up on artificial foods.

I think, therefore, that the proofs that scurvy is a condition of chronic ptomaine poisoning are quite sufficient to direct us in practice to an unfailing prophylaxis.

MR. GOADBY: There is one point that I think so far has not been considered in the discussion. The reader of the Paper finishes his remarks with the suggestion that scurvy is not due to the presence or absence of any particular food, or component of food, but rather to a primary mouth infection.

The discussion of the bacteriology of scurvy I am unable to enter upon, as so far I have had no personal experience with the disease. It is a disease that rarely comes under one's notice now, and during the last two years I have not seen a case at either the Dreadnought or the Royal Albert Dock Hospitals, although twenty years ago it was common at the Dreadnought.

There is, however, a condition of the mouth not by any

means uncommon in this country: one that is associated with general disturbance of health, often grave disturbance; and is, moreover, a condition which is apparently associated with direct bacterial infection and intoxication. It, moreover, bears some points of similarity to the mouth symptoms described in the Paper, particularly the character of the pus. As I have already said, I have no personal knowledge of scurvy, and I do not, therefore, wish to be hypercritical; but I should like to point out that a variety of this condition of the mouth, sometimes known as *Pyorrhœa alveolaris*, exists in an endemic form in the native of Western and Central Africa, as well as in India: more particularly in the gaols, as well as in Cuba and the Philippines; it, moreover, at times becomes epidemic, as it did among the American soldiers in Cuba.

The accounts given by Dr. Coplans of many of these cases, as well as the rapidity with which they responded to antiseptic treatment, makes it worth while to consider the possibility of *Pyorrhœa alveolaris*. I did not gather if any cases died.

DR. COPLANS: Twelve per cent. died.

MR. GOADBY: *Pyorrhœa alveolaris*, although common in this country, does not infrequently cause death; and I have, moreover, seen cases with purpuric eruptions, joint affections, and nervous symptoms. Cultivations made from these cases frequently produce death when injected into guinea-pigs, while the broth cultures filtered through a Pasteur-Chamberland filter often produce death on injection, with hæmorrhages in the supra-renal capsules. The ordinary pus cocci are not invariably present in these acute cases of *pyorrhœa*; neither is it a common thing to find them *post-mortem* in injected animals.

I should, therefore, like to ask the reader of the Paper if he has considered the question of *Pyorrhœa alveolaris?*

DR. NEWTON PITT: I am very much interested in this Paper of Dr. Coplans', and would congratulate him upon it, but I do not think he gives us enough facts upon which to form a conclusion as to whether infection occurred in his cases. It seems to me that if we are to accept his view that it is an infective process, we want to know whether it ever spread from mother to child; whether the cases were aggregated in certain tents; because in Russia, where scurvy is prevalent, there is no evidence that a child is ever infected by scurvy from its mother; and, generally, we

want more minute details as to the distribution of the cases, to enable us to form an opinion as to the mode of propagation.

I must say his explanation does not commend itself to me; and I am struck by the fact that the scavengers and muleteers, who probably had very hard work, were particularly affected; and it seems that with the dietary given, which was very insufficient, they would be the men most likely to suffer, because they had to do the most work.

The variations in the dietary are curious: for instance, in Group G, the natives received weekly as much as 7 lbs. of fresh meat, whereas our own troops and others got a very small ration. I understand from Dr. Coplans that this was partly due to the fact that one was under civilian and the other under military control.

Dr. Coplans says we must pay no attention to our previous experience, we must start afresh; but surely, when a new theory is started, we must consider it in the light of our previous experience. I should like to ask, for instance, how this theory would explain the fact that in 1780 there were as many as 1,400 cases admitted at Haslar Hospital; but after the introduction of lime juice, in 1795, into the Navy diet, there was one case in the year 1806, and also in 1807 only one case. The introduction of lime juice made this immense difference within ten years. At the "Dreadnought," in 1868, the number of admissions annually averaged 85, because the merchant service was not compelled, till 1865, to use lime juice; but in 1898 the average was 1-4. It would seem that if Dr. Coplans' theory is to hold, it must offer an explanation of numerous facts such as these.

Then again, at the Penitentiary, at Millbank, it was observed by Dr. W. Baly that men on short sentences frequently developed scurvy, while those on long sentences did not. On inquiry, it was found that the latter had potatoes in their dietary, which the former had not. On the introduction of potatoes into the dietary of the former, the occurrence of scurvy ceased.

Simply putting those two cases, how would Dr. Coplans' theory explain them? Professor Wright's Paper in the Army Medical Report for 1895, has struck me as being the one which brought most satisfaction to my mind.

There has always being a difficulty in explaining the cause of infantile scurvy, because my experience has been that it is only the children of excessively careful people who succeed in getting infantile scurvy. I have several times

been called to see a child who has been most carefully brought up, so far as cleanliness and hygiene are concerned, who has been fed on nothing but sterilised food, and who, I consider, has therefore developed scurvy; while this does happen to the children of careless people.

Professor Torup's theory of ptomaine poisoning is not sufficient to explain the facts of scurvy, and the evidence is more strongly in favour of an acid intoxication. Still, there are many other factors, and Dr. Coplans has done great service in bringing forward these facts. Though he does not bring forward sufficient evidence to upset the view which Professor Wright put forward a few years ago, the explanation of his own cases has yet to be shown.

PROFESSOR WRIGHT; I should like to say a word or two as to preserved milk. When we boil a faintly-acid urine we drive off the CO_2, render our urine alkaline, and then get a precipitate of alkaline phosphates. I take it precisely the same thing happens when milk is boiled. We get, then, it is to be assumed, a precipitate of the alkaline phosphates of lime. The evidence that this is so is indirect, but I think conclusive. When rennet is introduced into unboiled milk, it causes it to set into a firm clot. When it is introduced into milk which has been boiled, we obtain only a loose clot. It would seem to follow that the milk ought to become more digestible; but it is, however, notorious that boiled milk gives as much trouble in the stomach of a child as unboiled milk. On experimenting on the subject, I found that the boiled milk sets quite as firmly as the unboiled milk when a little hydrochloric acid has been added. Remembering what occurs in urine, I take it this is evidence that alkaline phosphates have been precipitated on boiling; and that if that boiling process was continued there would be thrown down all the alkaline phosphate of lime from the milk. Milk thus treated would therefore, I take it, be more likely to give acid intoxication than milk taken in its original condition.

In conclusion, I may advert to the question of the anti-scorbutic action of fresh as compared with preserved or salted meat. Fresh meat is meat with the blood in it. Salt meat is meat which has given up the mineral constituents of the blood to the leys. Herein lies the difference in blood in an alkaline food-stuff and an anti-scorbutic. We are told, for instance, that the Samoyedes find a remedy for scurvy by opening a vein of a reindeer and drinking the blood.

DR. J. T. C. NASH: The bacteria associated with the *gingivitis* in the cases alluded to in Dr. Coplans' Paper might easily be acid-producers, capable of forming enough acid to diminish the alkalinity of the blood, to the extent to which Professor Wright (as I understand him) insists upon, as the essential pathological feature in the etiology of scurvy.

The success of Dr. Coplans' method of preventive treatment by means of antiseptics to the mouth might easily be explained thus: the acid-producing bacteria become inhibited or destroyed, and consequently the commencing diminution of the alkalinity of the blood ceases, and further, consequently, the condition does not go on to scurvy proper.

It seems to me, from this view-point, Dr. Coplans' advocacy of antiseptic treatment is truly rational and preventive.

DR. BULSTRODE: There is one observation that I should like to make. No one has so far mentioned the fact that at least two bacteriologists have claimed to have found the specific organism of scurvy, and that other observers have regarded scurvy as being a communicable disease. Professor Wright referred to the researches and observations of Dr. Lind; and, if I may, I will read an extract which I have here, from a Paper read before this Society twenty years ago by Sir William Smart, R.N., Inspector-General of the Royal Navy, and in which he refers to the observations of Lind:*

Sir William observes: "As to the etiology of the disease, this great authority (Lind) discarded the prevailing theories that the salted meats of seamen was a direct cause, *further than their being less digestible, and therefore less nutritious;* and he taught that the want of vegetable food was not alone its cause. His views of the predisposing and exciting causes are summed up in these words, at page 516: 'I am fully convinced in my opinion that whatever weakens the constitution, and especially the organs of digestion, may serve, without any other cause, to introduce the disease in a slighter or higher degree, even among such as live upon fresh garden vegetables or the most wholesome diet in the open air.' These are important words from the pen of a physician so experienced as Dr. Lind was in this

* *Transactions of the Epidemiological Society of London.* New Series, vol. iii Session 1883-1884. Published by Messrs. Williams and Norgate, Henrietta Street, Covent Garden, W.C.

disease: *that more than the privation of vegetables is required to produce scurvy.*

"When it appeared in England, Scotland, and Ireland in the spring of 1847-8-9, it was attributed to the potato blight of the preceding years, and consequent privation of vegetable food of the lower orders; but it must be recollected that, especially in Ireland, a general state of famine prevailed among the poor, a want of every kind of wholesome food, besides having the standard vegetable supply in great deficiency, as well as defective. Lind states 'that he had known seamen's messes in which vegetable food was not used above twice or thrice a month during three years, and yet there was no scurvy.' 'That in 1759-60, there were two frigates employed in conveying merchant ships between Plymouth and the Downs, touching at Portsmouth, being seldom above forty-eight hours at sea, and during twelve months their crews had not any salt provisions oftener than once or twice a fortnight; but notwithstanding the constant use of fresh beef and vegetables, greens, etc., the companies of both ships became greatly afflicted with scurvy' (vol. v, p. 27). 'That now in July, 1771. I have under my care several men very ill of the scurvy, sent from the guardships at Spithead, which ships have never been at sea. These men, being pressed into the service six or seven months since, and not permitted to come on shore, solely from confinement on board ship became highly scorbutic' (vol. v, p. 519). 'That Porchester Castle being full of French prisoners in 1760, hulks were employed, in which they had the same rations of beef, fresh bread, and beer, etc., as those within the castle walls, with an open quadrangle; but these remained free from scurvy, while those cooped up between decks became highly scorbutic.' Other striking instances could be cited to show that something beyond diet alone is required to generate the scorbutic diathesis. And what is that something? To which the safe reply is: 'Not yet sufficiently known; but usually multiple.'"

It appears to me that this quotation is very interesting and pertinent in relation to the conclusions arrived at by Dr. Coplans. Of course, it may possibly be that the disease that Dr. Coplans has brought before us is not scurvy, although it is very much akin to the symptoms which are generally regarded as in the aggregate making up what we term scurvy. But I think, if we look at it as a separate entity—and I do not see why the past view should prejudice our opinion—we must come to the conclusion that the orthodox theory does not explain many of

the facts which Dr. Coplans has presented to us, and for this reason his Paper must be regarded as a valuable contribution to the subject. Probably the author would not himself contend that his facts are sufficient to upset other well-established facts as regards the etiology of conditions called scurvy. It may be that, as Lind has suggested, there may be several causes, if not of scurvy, of something which is not easily distinguishable from it.

THE PRESIDENT: I only want to suggest one thing—that we should tender to Dr. Coplans our grateful thanks for his Paper, and for the discussion to which it has given rise. (Applause.) I will now ask him to reply to the criticisms upon his Paper.

DR. COPLANS, in reply, said: I wish to thank you for listening to me so patiently. Professor Wright and Dr. Pitt have raised the question of the identity of infantile scurvy with scurvy in adults. I understand that infantile scurvy yields readily to such treatment as fresh meat and its juice; but you have ample evidence here that scurvy arose in some units, despite the large quantity of fresh meat consumed. I do not think, therefore, that these two conditions are identical. Dr. Pitt has asked for evidence as to transmission of the disease from mother to child. Such information I cannot give him, because these cases of scurvy were chiefly among men and boys. I am quite sure that at the time I was at Standerton the food was uniformly excellent; and both from personal observations and from the observation of those in charge of the men, I made certain that the food was being consumed. Yet it was at this time that scurvy broke out. Colonel Lane-Notter tells you that, during his time at Standerton, food was bad; but during his time there was no scurvy. Professor Wright points to scurvy as a disease in which there is reduction of alkalinity of the blood; but there are other diseases in which there is an acid intoxication and reduced alkalinity of the blood. Professor Wright says that when one camp shows many cases of scurvy and another few, it might be due to the presence of overt cases in one camp and not in the other. Why should all the overt cases occur in one or two camps—that of the Native Sanitary Corps and the Repatriation Department respectively? He says that lime juice contains citric acid, and is of no use whatever from the point of keeping off scurvy; Dr. Pitt, on the contrary, states that after the introduction of lime juice into

the Navy, scurvy practically disappeared. I do not see that the lime juice has any bearing on the matter: but that the prevention of the spread of scurvy is due to the greater cleanliness and the generally improved standard of hygiene which has crept over the Navy—as well as other sections of the community—during the past century. To Major Firth I wish to reply that the absolute quality and freshness of the food is quite beside the main contention of the Paper: since whatever the standard of the food, it would affect all the communities equally. He does not think the Native Labour Corps and the Scavengers had sufficient food for their work. Here again I can only say that the natives had enough and to spare. They put aside their spare mealies, and fermented them into a spirit called "mahow." The natives said they had as much food as they wanted. Their overseers said the same thing. The Repatriation Department natives, on the other hand, had too much meat; and the question debated by their medical officer and the magistrate was not "what to add," but rather, "what to take away," on account of the obviously wilful waste of food that went on. I do not understand how Major Firth obtained an acid ash by incinerating food.

Colonel Lane Notter has drawn your attention to the insanitary condition of Standerton. I took occasion, in May, 1903, to publish in the *Journal of Tropical Medicine*, some notes on the insanitary condition of Standerton, and what Colonel Notter describes is but a tithe of what occurred at this dreadful place. He has drawn your attention to the foul-eating habits of the natives generally. I do not think the native is any better or worse than those people in other countries who eat "high" game, or entrails of animals, though the subsequent dishes be called by French names. He takes exception to the theory I put forward, in that if there were a bacterial infection, no section would be entirely free. Is this a just inference? How often is a typhoid outbreak localised? Could the fact that during 1903 not a single Jew in London died of small-pox—though Jews form 3 per cent. of the entire population—have any bearing on the general infectivity and high mortality-rate of small-pox?

Colonel Notter says that while he was at Standerton the food was bad. The mealies were mouldy—of the South African sun-dried variety—but there was no scurvy. After his departure, the mealies supplied to natives were very excellent—of the South American kiln-dried variety—and

then there was scurvy. I am well aware that at one time Major Beveridge found, in the Orange River Colony maize, the organisms causing *pellagra*.

Colonel Davis and Dr. Bulstrode have narrated to you instances of outbreaks of scurvy when there was no deficiency of food or lack of vegetables in the dietary. In the one case, the trouble was aggravated during the rains, and when the men got out into the open for exercise, after the rains ceased, scurvy disappeared. Similarly at Standerton: scurvy became most prevalent during the rainy season, but its disappearance might also be attributed at Standerton to departure of large bodies of natives from the scene after the rains. Colonel Davis, with reference to the French soldiers in the Crimea developing scurvy during the second year of the campaign, and not in the first year—in contradistinction to the British troops' experience—is good enough to draw the conclusion that the French hygienic conditions were not so good in the second winter as in the first; and thereby I take it his conclusion supports my contention. During their first year the French went foraging for rations —French soldiers are very enterprising—and their morale was excellent, their standard of cleanliness was of a high order, and they took much open-air exercise. Later on, they gave way before the difficulties of the campaign: their morale depreciated, cleanliness was less apparent, and they succumbed to scurvy. The English troops, however, as time went on, were better housed, better fed, and less overcome by the difficulties of the campaign.

Colonel Davis says scurvy is not present in Madras; and another says it is endemic in European Russia—but the habits of their natives have not being indicated. Professor Wright noted that the Samoyedes know the anti-scorbutic value of blood, and drink it fresh from the reindeer. In return, I wish to point out that Jews all over the world bleed their animals to death, and take extraordinary care that they shall not consume the blood—actually dissecting blood vessels out of the flesh before cooking, the process being termed "porging." This is in accordance with the Rabbinical code.

Dr. Pitt says short-sentence people in a certain prison contracted scurvy, but not the long-sentence people. He mentions potatoes as the sole difference, but makes no mention of the comparative confinement in each case, the degree of congregation of prisoners, the open-air exercise, and the general hygienic surroundings.

I wish to point out to Dr. Willoughby that the quality

of food supplied to natives during the outbreak was uniformly excellent.

Professor Wright spoke of overt and suppressed scurvy, and said that overt scurvy takes time to develop. Will he or Dr. Pitt explain why that unit, the Native Sanitary Corps, with easy working hours, with work light though disagreeable, with the shortest period of military service, showed the heaviest incidence of overt scurvy?

Dr. Goadby has spoken of *Pyorrhœa alveolaris*—but is this disease primarily fatal, or has it a mortality rate approaching 12 per cent? This disease is said by him to have an association with insufficient food and generally depressed conditions; but in the outbreak of scurvy at Standerton, such conditions were absent. His description of some of his cases of *Pyorrhœa* would lead one to suspect that they were cases of scurvy.

We had natives with us from all parts of Southern Africa; and I cannot say that scurvy was more noticeable among those who came from malarial districts than among others from non-malarial districts.

I have quoted you instances of isolated units, tents, and individuals respectively, being wholly free of the disease, while in other instances few escaped. *They were all subsisting on a dietary uniform at any one time.*

In any camp, with an average of nine individuals per tent, it is an extremely difficult undertaking to allocate any tent solely to the use of any particular set of individuals, and subsequently to maintain that status, especially having regard to the free-and-easy intercourse between individuals that characterises camp life. Statistics based on the incidence of scurvy per tent in such camps can be of little real value, and were not gathered in consequence.

I venture to recall to you the value of mouth inspection, mouth antisepsis, rest in the open air, coupled with measures of isolation, adopted in connection with these cases. I trust that these notes will throw some light on many of the obscure points of this Paper.

SOME NOTES ON PYORRHŒA ALVEOLARIS.

Bödeker's "Dental Diseases," 1894.

Pyorrhœa alveolaris (synonyms, Rigg's disease. Chronic purulent pericementitis; pyorrheo-alveolo (of Toirae); Cemento-periostitis (Magilot); Gingivitis expulsiva (de Calve); Alveolitis infectosa (A. Witzel); Blenorrhœ der alveolen (Steinberger).

Said by Pierce, at New York Odontological Society meeting, 1894, to

be but a symptom of gout. Others oppose this on ground that uric acid may exist in system without this condition, and that uric acid diathesis exerts no more influence in this direction than does consumption, anæmia, nephritis, hepatitis, diabetes, arthritis, syphilis, typhoid, and chronic diseases of the nervous system, etc., which are associated with the disease. Many cases of the disease can be attributed to nothing but heredity.

Sewell's "Dental Surgery," 1901, pages 427, etc.

Pyorrhœa alveolaris is defined as the condition of slow wasting of the alveoli, gradual loosening and shedding of the teeth, attended by discharge of pus, generally small in quantity, from within the free margin of the gum and alveolus, and with deposit of tartar, mostly in form of small isolated hard nodules, upon the denuded surface of the teeth.

The disease is usually extremely chronic, months elapsing between shedding of individual teeth, and from five to ten years pass before whole set is destroyed.

The process commences in the lower incisors usually, and gradually affects other teeth of the jaw, not necessarily those adjacent.

The symptoms commence with a spongy, slightly swollen condition of the free edge of the gum. The gums recede, and the recession is often followed by deposit of tartar upon the denuded surfaces of the teeth. Slight inflammation of the peri-dental membrane is next recognised, and this swollen membrane raises and loosens the tooth in its socket, and a probe can be passed along to the root. From these pockets constant discharges of foul-smelling pus or muco-purulent fluid take place, and the discharge may become profuse. Within the gum pockets there are usually to be discovered small nodules of dense matter, dark in colour, scattered over the denuded surface. The alveoli waste rapidly, and the teeth may become so loose as to fall out in mastication.

Pyorrhœa alveolaris is the opprobrium of dental surgery.

Etiology.—Virtually nothing is known.

Pathology.—Little known.

Prognosis.—Always unfavourable; once established, the disease progresses in spite of treatment until the whole dentition is destroyed.

No organic or functional disorder is known to give rise to the disease. It occurs in many patients whose care of the mouth and teeth amounts to fastidiousness. It is a disease unknown in the young; it is a disease essentially of middle and advanced age.

When the affected tooth is extracted, the discharge from that spot ceases, the alveolus assumes a healthy condition, and the gum heals normally. The extracted tooth when examined yields negative results; on its surface may be a few nodules of tartar, and later, the peri-dental membrane may be thickened.

Pathologically, the disease may be compared with sycosis, to which it bears at least a superficial resemblance.

The *Bacteriology* presents no features differentiating the disease from other purulent discharges. Several varieties of pyogenic cocci usually discovered.

The *Diagnosis.*—Appearances are characteristic; barely mistakable. May be simulated by early shedding of the teeth—but in this there is no inflammation—or by injury to the teeth and jaw, with chronic inflammation, when a history of violent injury is obtainable.

The *Treatment* consists of assiduous antisepsis of the mouth and removal of the tartar, and is merely a temporary check on the advance of the disease.

Special frames for carrying teeth to replace those lost are recommended.

The disease terminates when all the teeth are lost, and does not of itself appear to be fatal.

Ill-health may occur in connection with the disease, and the disturbance is more noticeable in sensitive patients, who are conscious of the foulness of their breath; and it is a question in long-standing cases which is the primary condition. The disease may follow long and wasting illness, such as typhoid fever. Careful inquiry and investigation in most cases has failed to discover any organic or functional disorder capable of giving rise to pyorrhœa.

THE EPIDEMIOLOGY OF ENTERIC FEVER AND CHOLERA IN HAMBURG.

By Dr. REINCKE, Medical Officer of Health of the City of Hamburg, and Corresponding Member of the Epidemiological Society of London.

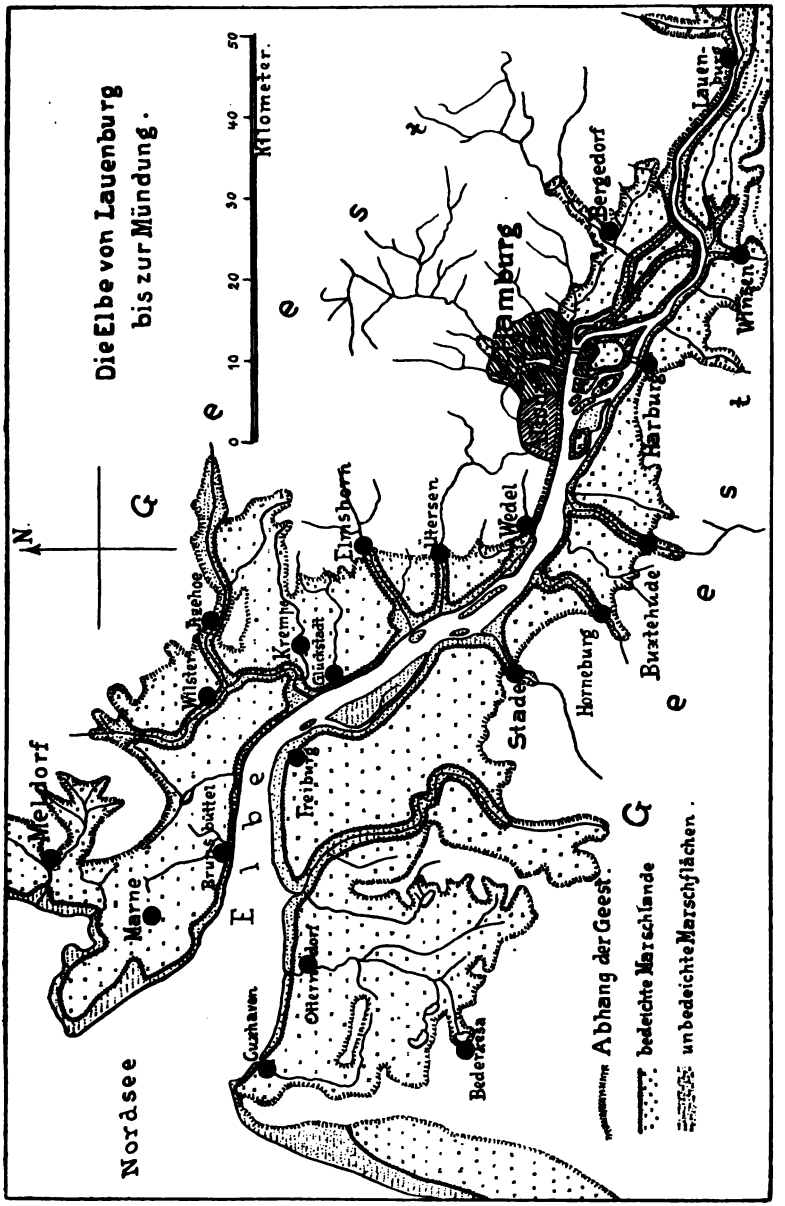
(*Read: March 18th*, 1904.)

In order to understand aright the epidemiology of enteric fever and cholera in Hamburg, it is necessary to have a clear picture in one's mind, not only of the topography of the town, but also of the topographical arrangement of the surrounding district; for the reason that the particular conditions of our locality are in a special manner favourable to the diffusion of enteric fever and cholera from the town to the country, and from the country to the town.

Hamburg, as you are aware, is situated in the great North German lowland, which is of diluvial origin, made up of glacial drift, sand, clay, gravel, and erratic boulders, scattered pell-mell, overlying the tertiary formation, and in many places rising like a plateau to a height of fifty metres and more above the level of the sea. This formation, which is, comparatively speaking, poor in water, is termed by us the "geest." At the sea-coast and mouths of rivers lies the alluvial formation, "the marsh," which extends in front of and beyond the "geest." It is, however, only met with in those parts where there is an ebb and flow, that is to say, only at the North Sea, not at the Baltic.

These marshes consist of the sediment deposited by large rivers, which empty themselves sluggishly into the sea by wide mouths. It is only through the activity of man in building "dikes" or embankments that they are made habitable and secure against floods. In some places they form islands between numerous water-courses; in others they form a wide boundary to the "geest," covering miles of flat expanse at sea bays and mouths of the river. Hence it is clear that the part of the estuary of the Elbe, as far as above Hamburg, once formed a deeply-indented bay (Fig. 1 gives a picture of these conditions).

Fig. 1.


Die Elbe von Lauenburg bis zur Mündung.
N.
0 10 20 30 40 50 Kilometer.
Nordsee
Meldorf
Marne
Brunsbüttel
Wilster
Itzehoe
Elbe
Krempe
Glückstadt
Elmshorn
Uetersen
Wedel
Hamburg
Bergedorf
Lauenburg
Winsen
Harburg
Stade
Horneburg
Buxtehude
Cuxhaven
Otterndorf
Bederkesa
Abhang der Geest.
bedeichte Marschlande
unbedeichte Marschflächen.

The marshes are very damp, and are intersected by numerous drains, which at low tide empty themselves into the river by means of sluices. A somewhat larger drain, which flows parallel with the embankment, goes by the name of the "Wetterung." The dwelling-houses lie on the inside of the embankment, between it and the "Wetterung," at irregular intervals, in even rows, extending for miles. The fertile land lying behind is used for feeding cattle and growing vegetables (see Fig. 2).*

As naturally follows from such an arrangement of the houses, all kinds of refuse from the dwellings, cattle-stables, and dunghills very easily finds its way into the "Wetterung," which at the same time is used for drinking and domestic purposes. For all water from the marsh wells contains so much iron that it cannot be used for drinking. And in cases where the "Wetterung is not used, water is taken from the river outside the embankment, which also cannot be regarded as free from pollution. Those houses are best off that collect rain-water from the roof in underground cisterns. You can understand that, under such circumstances, grave insanitary conditions easily arise, although the people in many of the marsh districts are often of almost "Dutch cleanliness." When enteric fever occurs in these districts, it can only be stamped out by slow degrees, and with much difficulty. In former times it never died out altogether. When the people of Schleswig-Holstein spoke of marsh fever, they usually meant enteric fever, and not malaria. Such infected marsh villages are at the present day also the source whence the disease is being continually reimported into the town, especially when these villages are close to a big town, such as Hamburg, with which they keep up regular intercourse in pursuing their market trade, bringing all kinds of goods to the town, more particularly articles of food which have come in contact with infected water in the "Wetterung," and are consumed in a raw state. Mention must, first of all, be made of milk, which is conveyed in vessels which have been previously cleansed and washed in marsh ditch water; further, different kinds of vegetables, salads, radishes, and fruit, which are probably stored in the drains (or ditches) to keep them fresh till market day.

In bringing forward these circumstances, it is, of course, not implied that the "geest" is altogether free from enteric fever. There, too, the disease may be imported, the wells

* Not here reproduced.

infected, and from them in its turn the milk also. Still, such cases are much rarer here, and when they occur are more localised, and therefore easier to suppress.

Moreover, in the course of time, decided improvements have been made in the marsh, particularly by the improvement of the water supply. This has been effected partly by learning and introducing methods to free the water of the different wells from its iron, thereby making it useable, partly by the establishment of central water supplies from deep-bore wells giving very good water. In addition to this, various precautionary measures in connection with the position of privies, dung-heaps, and slaughter-houses, with the handling of milk and other articles of food, have contributed to the improvement of the sanitary conditions of the district. Still, there can be no doubt that the marsh villages, in spite of all precaution, are continually running risks, and are at the same time a constant source of danger to the town.

I believe that in this country, also, you have small districts at the mouths of the Humber, Trent, Ouse, etc., where the conditions are similar. Dr. Reece, in a report of August, 1903, to the Local Government Board, described the sanitary conditions in Thorne Rural District exactly as we know them to be with us in our marshes. This state of things on the Continent is not confined to the neighbourhood of Hamburg, but exists also along the whole of the North Sea coast, from Schleswig as far as Holland and Belgium.

And with slight alterations, the conditions in the towns situated in the marsh are the same as those in the country districts. Hamburg was founded by Charles the Great as an advanced seat for a bishopric among the heathen, and was built on an elevation in the "geest," or high land, between marshy plains which were not banked in. But so soon as temporal interests became more and more important (together with the spiritual interests), the buildings extended into the marshes. And the more the population turned its attention to commerce and shipping, the more did the town extend, in the direction of the stream, into the marshes. Thus, at the present day, a considerable portion of the town lies in the marsh, where houses may only be built after numerous props have been driven deep into the ground for support. These marsh portions of the town have developed from banked-in marsh islands called "Polder," and they are still intersected by numerous water-courses, which in Hamburg go by the name of "fleete." They represent exactly the same thing as the "grachten" in Dutch towns

or the canals in Venice, with the only difference that in the latter town there is no flowing fresh water, but only the salt water of the Adriatic. These Hamburg navigable canals in former times received all refuse from the houses abutting on them, and at the same time served as sources of water supply. We have still old pictures showing this state of things. It is therefore pretty certain that the sanitary conditions in the town were formerly very unfavourable, although definite details are wanting. The danger was not suspected, and the value of cleanliness was not recognised till the year 1831, when cholera came and opened our eyes, as well as those of the whole world, to the danger. It was then that your country took the lead, showing a good example, and teaching how towns might be kept sanitary by means of good drainage and good water-supply; and Hamburg was the first Continental town to profit by your English experiences. In 1842 our town was visited with a devastating fire, lasting three days, laying in ruins a very considerable portion of it. It was then that we turned to your countryman Lindley (who died only lately, at a good old age), and he provided, not only the newly-built parts of the town but also the remaining old parts of it spared by the fire, with a general system of sewerage and a central water-supply, both of which were completed towards the end of the 'forties. This was the beginning of improvement in the health of the city; and I will show you later, from the curves of enteric fever and cholera, how favourably both measures have acted, in that they not only improved the drinking water, but also led to a greater cleanliness in every respect: in house and yard, in kitchen and cellar, in clothing and care of person. We can thus account for the steady decline of tuberculosis in Hamburg since the great fire, to which fact Dr. Bulstrode recently drew public attention. Unfortunately, this steady progress in sanitation has not continued, because, as is well known to you—and as I am just going to show you in detail—the central water-supply had serious shortcomings.

Before entering on this important part of the subject, I should like to speak shortly of the shipping conditions, which greatly resemble the conditions in the country districts described above. In so doing, I will except the sea-going vessels which, as a rule, have good drinking-water on board, and whose passengers and crew do not easily come in contact with the river-water. But it is quite another thing with the river craft, which come into Hamburg in great numbers from the wide river-path of the Elbe, and

from the Oder and Vistula, which are connected with the Elbe by means of canals. There are often as many as 800 and more of such large river-craft in the harbour, each of which is manned by a family—a skipper with his wife and children, and one or more hands. In spite of all precautionary measures and regulations on the part of the authorities, those on board allow their dejections to go into the river from which they at the same time draw their drinking-water. Although the habits and mode of living of these people are gradually improving, yet they still leave much to be desired, and you will understand of what importance it is to a town for the epidemiology of enteric fever and cholera, when a very great number of such ships lie close together in its harbours. Naturally, similar dangers arise from the still more numerous small tugs, lighters, barges and boats, and from the numerous businesses and occupations carried on at the water's edge. Nevertheless, these take a secondary place in comparison with the dangers which arise from the river-craft, because in all those other cases only men are occupied, and that, too, only during the daytime, whereas here women and children are also concerned, who have less discernment, and who live day and night on the water, and can easily, unperceived, take advantage of early morning and late evening to pollute the river.

After this digression, I return to the subject of the water-service.

The water for the central service was from the very beginning taken from above the town, where it was stored but not filtered. This was a marked improvement on the previous state of affairs; as at low tide the fresh water from the upper course of the river was taken in; and as even in high tide the river at this spot was not nearly so polluted as near the town and in the water-courses of the town, where it receives the unpurified sewage of the whole city. Nevertheless, the innate danger consisted in this: that in the case of a low level of water in times of drought, or in the case of spring tides raised higher by the winds, the tidal wave, which carries with it all refuse out of the harbours and sewers, can come much higher up-stream than is usually the case. Consequently, part of the polluted material from the town, although in a modified or rarefied form, might reach the intake and enter the pipes. This danger became gradually greater and greater, although the intake was taken higher up the stream. The town at the time of the fire of 1842 numbered about 160,000 inhabitants; in 1884 about 440,000; and in 1903, 740,000. In consequence, as

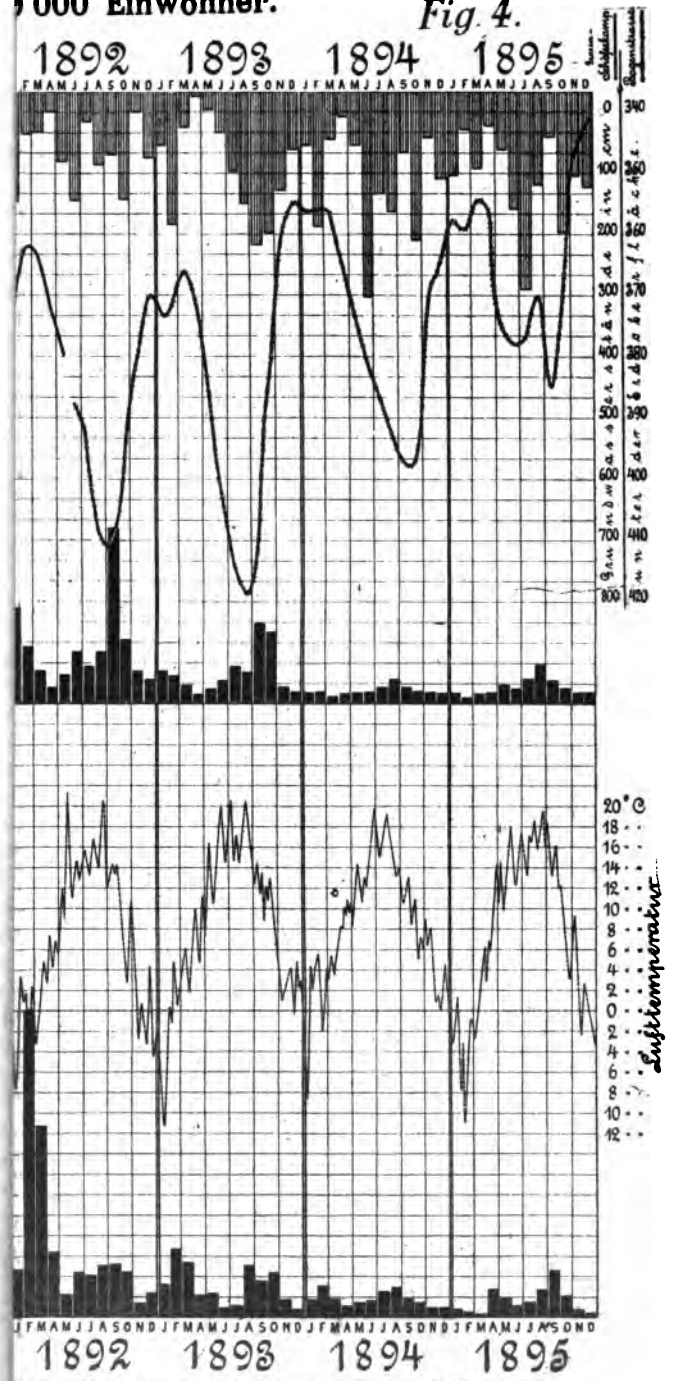
time went on, more and more sewage was emptied into the Elbe; the town buildings and harbours extended farther up the stream, the shipping increased year by year, and regulation works were built, the object of which was to deepen the water-way, and bring more water into the harbour for ships with a deeper draught. All these circumstances contributed to allowing sewage more easily and more frequently to make its way to the intake. Not till the introduction of the central sand filtration, in 1893, were all these dangers at an end. I will not take up your valuable time with the details of the development of this state of things, which passed through many stages and involved many battles; nor will I trouble you with the proposals to remove the intake still higher up-stream. It might not even interest you. For the present purpose, you have enough with what I have told you, and with that which you can deduce for yourselves from the plan (Fig. 3). I should only like to add that the area of the piping of the water-service coincides with the political area of the town. The political boundaries coincide exactly with the boundaries of the water-service. On the East, the Prussian town of Wandsbek, which at the present time has about 30,000 inhabitants, joins on to Hamburg without any visible line of demarcation. This town of Wandsbek has waterworks of its own, supplied from inland lakes in Holstein. On the west of Hamburg lies the Prussian town of Altona, with, at the present time, about 168,000 inhabitants, and which, like Hamburg, gets its water from the Elbe. The waterworks which were built by your fellow-countryman, Thomas Hawksley, during the years 1857-59, lie ten kilometres below the town, and take, therefore, the river-water polluted by the sewage from both large towns —Hamburg and Altona. Since the opening of the works in 1859, the water has been carefully filtered. Pipes from these works supply the town of Altona almost as far as the political boundaries of Hamburg. In Fig. 3 these boundary lines are shown very distinctly. It is interesting to observe how first one and then the other of the two water-services was infected with enteric fever. In the case of the Hamburg service, it might happen any moment; in the case of the Altona service, only when the filters were found wanting, or when unfiltered water was pumped into the mains. Each of these mishaps occurred often during the first few years of working, because the technical detail of filtering had first of all to be learnt, and then put into practice, and also because, acting without knowledge of the hygienic dangers of unfiltered water from the Elbe, no care

was taken to avoid supplying unfiltered water when repairs were being executed. At that time, in the filtration of water, attention was paid chiefly to the æsthetic purifying of it alone. In subsequent years, all cases of failure in the efficacy of the filters are in all probability to be regarded as due to frost in hard winters. The water in the open filters freezes, and the coating of ice makes it impossible to cleanse the top layers of the filtering sand, as is otherwise done at regular intervals by carefully removing the slimy layer which has been deposited on the sand, and which in time stops up the pores of the filter. When a hard frost came, as long as the better modern methods were unknown, the authorities were obliged to use all kinds of extreme measures to make the filters pervious, and very often the function of them was heavily damaged by those measures.

Here, in Fig. 4, I show you two diagrams which represent the number of monthly attacks of enteric fever in Hamburg and Altona for the years 1883-85, calculated per 1,000 inhabitants. During these years we had in Hamburg different large epidemics of enteric fever, which you will see occurred as a rule in autumn, if not in winter itself; and they repeatedly overlapped into the following year. Altona did not share in these epidemics, at the most only in so far as is easily explained by the close intercourse between the inhabitants of the two towns. But when the epidemic in Hamburg receded it began to increase in Altona (but much more severe, much more sudden, and rising to greater heights than in Hamburg), only to fall again very rapidly. Sometimes an epidemic would break out in Altona, when none preceded it in Hamburg. This happened in the beginning of 1891 and 1892, while, *vice versâ*, there were epidemics in Hamburg which left Altona untouched: as, for example, in the autumns of 1889 and 1892. It cannot, therefore, be assumed that enteric fever always broke out in the low-lying Altona, whenever Hamburg sent an unusually great number of enteric bacteria through the sewers into the Elbe. But we must conclude that the part of the Elbe running past these two towns was in a constant state of infection, and that enteric fever broke out in Altona when the filters did not properly perform their functions, owing to hard frost in January and February. On the other hand, outbreaks of enteric fever occurred in Hamburg when the water in the Elbe was low, in the autumn of dry years, which led to the tidal wave coming unusually far up the river, bringing with it, as far as the intake of the town waterworks, a large quantity of Elbe water polluted by

) 000 Einwohner.

Fig. 4.



eines Brunnens an der Bogenstrasse 1892 bis 1895.

ler Lufttemperatur 1883 bis 1895.

enteric discharges. Such dry seasons occurred in the years 1885, 1886, 1887, and 1892; while, on the other hand, the dry season of 1883 brought with it no appreciable increase of enteric cases, for the reason that at that time an alteration of the river bed, which was to shorten the path of the tidal-wave, was not yet completed.

Only by these circumstances can we explain how it is that in these two towns, merging into one another without any definite line of demarcation, enteric fever ran such different courses. The epidemic in Hamburg, with a gradual rise and gradual fall, lasted nearly eight months; in Altona, with the exception of the winter 1887-88, the epidemic, in the form of severe explosions, lasted only one or two months, and was at its height at a period several months later than in Hamburg. But in both towns the whole area of the water-service was always attacked practically in the same way. From the drawing in Fig. 5, which represents the state of things in the winter 1887-88, you will see how matters stood within the borders of Hamburg and Altona. On the left lies Altona territory, on the right Hamburg territory, separated by the characteristic boundary line. Below, the Elbe is indicated. Each of the twelve areas represents the state of affairs in series of monthly averages. You will see that in July, 1887, and June, 1888, at the beginning and end of the series, on both sides alike there was but little enteric fever: only two to five cases per 10,000 inhabitants. Matters stand thus in Altona up to November, whereas in Hamburg the number of cases goes on increasing, till in December it is at its height—26 per 10,000 inhabitants. Then comes the decrease, so that by March the same conditions are reached as in the preceding July. The very month Hamburg is free from enteric fever, Altona is attacked most severely; likewise, with 26 cases per 10,000 inhabitants, the increase in the number of cases beginning in the preceding November. And exactly the same thing occurred in other winters, up to 1893. Since then the central system of filtration has been introduced in Hamburg; and here, as well as in Altona, we have learnt to master the difficulties during the period of hard frost. Since that time, large epidemics of enteric fever have not occurred in either town.

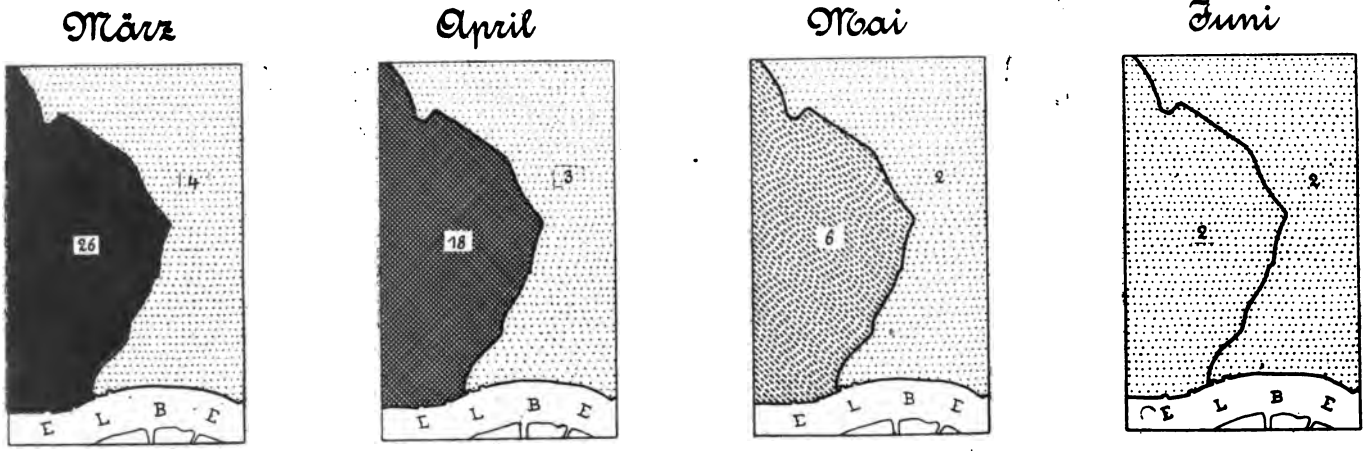
But isolated cases of enteric fever have remained, viz.:—about three to four per annum per 10,000 inhabitants, 200 to 300 cases, with, approximately, thirty deaths. Among these there may be many cases that are wrongly diagnosed as enteric fever, as diagnosis is not always very easy; and

young medical men have no longer much opportunity of becoming acquainted with the disease. On the other hand, many slight cases may escape detection, which are of no clinical interest, and which are not recognised as enteric fever, but which may nevertheless have been important factors in the spread of the disease.

Thus the number of reported cases may be approximately correct. Nearly a third of these can, as a rule, be shown to be imported, chiefly from North American ports, and from small villages in the mountains or by the sea, where families go to recruit during the sommer holidays, never heeding that in many of these places the sanitary conditions leave much to be desired. Another considerable series of cases can be directly traced to the Elbe; they concern skippers and boatmen on the river-craft in the Elbe, or workmen who work at the water-side. Then we have had, in some years, groups of cases which with more or less probability could be traced to infected milk from a particular milk-service, or from one particular farm in the neighbourhood. Lastly, there are always cases which are obviously directly infected—for example, among sisters and brothers in small dwellings, or among nurses in hospitals. After subtracting all these cases, there still remain 100 or even more which cannot be traced to any known source of infection. Naturally, however, one can easily content oneself with the assumption that, considering the intercourse between the inhabitants of a large city, nothing is to be wondered at. The incubation period is two to three weeks; so how can the person always remember how, where, or when he may possibly have exposed himself to infection, or even judge whether he was in danger of infection or no; as, for example, when bathing in the Elbe, visiting such and such a house, or partaking of food and drink which, perhaps, came from a distance from an infected place, as in the case of oysters? But one may also ask oneself the question that has been asked by more than one person, whether these few cases cannot be traced to the filtered Elbe water, as the filters cannot be absolutely relied upon to keep back every germ. Sometimes it appears as if the number of cases of enteric fever in the town increased after the filtered water showed for a time an increased percentage of germs, just as is believed to have been observed here in London. Sometimes, however, the comparison does not hold good. In my opinion (at any rate with us), this question cannot be decided statistically, because the numbers involved are too small. The matter will not be solved till we have deep

Typhuserkrankungen

Fig 5.



well-water instead of filtered Elbe water in our water-mains. Decisive steps towards this alteration in the water-supply have already been taken, and in two years' time we hope to be able to supply a third of our needs with deep well-water. And I do not doubt that after this is accomplished, the remaining two-thirds will follow in the not-too-distant future. What enteric fever then remains will certainly have nothing to do with the water-supply. Already now I should like to remark that several large towns of Germany, supplied with deep well-water, have not less enteric fever than Hamburg.

After setting forth these considerations, I will here show you, in Fig. 6, a diagram representing the yearly number of deaths from enteric fever, calculated per 10,000 population, since the year 1840 up to the present time. I have omitted the figures for preceding years, as they are too unreliable to be of any value. The number of fatal cases, however, must be taken instead of the actual number of cases, as we only know the latter since the year 1872. In this diagram you will see that, since the great fire of 1842, the prevalence of the disease has steadily diminished, although interrupted by great fluctuations; and, further, that since 1894 the number of cases has been very few indeed. This favourable condition of things since 1894, and the gradual sinking of the curve since 1892, can be easily understood without further statement, after what I have shown you up to this point. But how are we to explain the great rising of the curve in 1857, 1865, and 1889?

This is a question on which many opinions have been expressed, without any completely conclusive answer being given.

In Fig. 6, over the columns representing the number of fatal cases of enteric fever per annum, I have given also the index of rainfall in millimetres, as far as these numbers are known; and in Fig. 4, which shows the number of monthly cases, I have given, in addition to the average temperature curve, the average level of the ground-water for each month. Comparing the two representations, you will see that the level of the ground-water and rainfall are practically parallel, that is to say, that the level of the ground-water falls as the rainfall decreases, and *vice versâ*. Further, you will see clearly that the great epidemics of enteric fever fall in dry years, when there is a low level of ground-water, whereas in very rainy years enteric fever obviously recedes. These observations agree in every respect with the celebrated observations of Petten-

kofer, of Munich. Explanation is as difficult as the facts are self-evident, because the Pettenkofer explanation of facts clearly does not hold good, after the importance of the part water plays in the diffusion of enteric fever, which was disputed by him, has now unquestionably been established. Most seductive of all is the assumption that dry seasons specially favour the transmission of enteric germs from the dejections of enteric cases to the alimentary canal of other persons. Without entering upon many self-evident general reflections, I may state that in the case of Hamburg this theory is supported by the fact that the explosive outbreaks in Altona occurred in the middle of the coldest part of the winter: a season which is usually regarded as being particularly free from enteric fever; and, further, these explosions obviously occurred because, through the sudden disturbance of the process of filtration, the hitherto impeded progress of the bacteria was made possible from the Elbe water (into which they had entered with the discharges of the patient by means of the sewage) to the alimentary canal of other persons. Is not the conclusion most obvious that similar mechanical circumstances also decided the course of the Hamburg epidemics? I have already tried to make this appear probable with regard to the epidemic of the 'eighties; and I do not doubt but that similar mechanical circumstances—that is to say, high floods and a low level of water in the Elbe, both of which are favourable to the pollution of the water-service with the contents of the sewers—had a decided influence on the epidemics of 1857 and 1865. But it is very difficult to sift such questions sufficiently thoroughly, because the average numbers referring to the monthly levels of the water of the Elbe, or the size of the flood, or the fatal cases of enteric fever, do not suffice to settle the matter. More must be known of the local spread of the disease in the town, of the different floods, and of the conditions of the stream at the time: facts which can no longer be ascertained. But the probability of the case seems to me to speak in favour of my theory, although it does not, of course, explain everything. I consider it a dangerous practice, and one to be avoided, to want to explain in a word the endless intricate workings of Nature. I should like to specially emphasise the fact that dry seasons, in some way or other, seem to particularly favour infection by contact, and the different indirect transmissions of infection in different houses, by means of articles of food, vegetables, or flies. For in many places where periodic epidemics of enteric fever show a similarity with

Hamburg, there is no tidal ebb and flow, and they cannot all be explained by the theory that dry years and seasons specially favour the pollution of wells. I should like to believe that in former times the latter circumstance played an important *rôle*, at all events in Munich. Whether, however, it can be applied in all cases, or whether altogether different circumstances do not come into consideration in the periodic outbreak of enteric fever, must remain an open question. In this respect we have still much to learn. In any case, you will see by my curves that since 1894, when Hamburg's sanitary condition was improved, fluctuations in the rainfall and level of the ground-water have no longer had any influence on the frequency of the outbreaks of enteric fever—just as is the case with Munich since the cesspools were either made impervious, or done away with altogether, and replaced by sewage which freed the numerous town wells from the pollution brought about by the pervious pebble subsoil from the privy-pits.

And now to cholera!

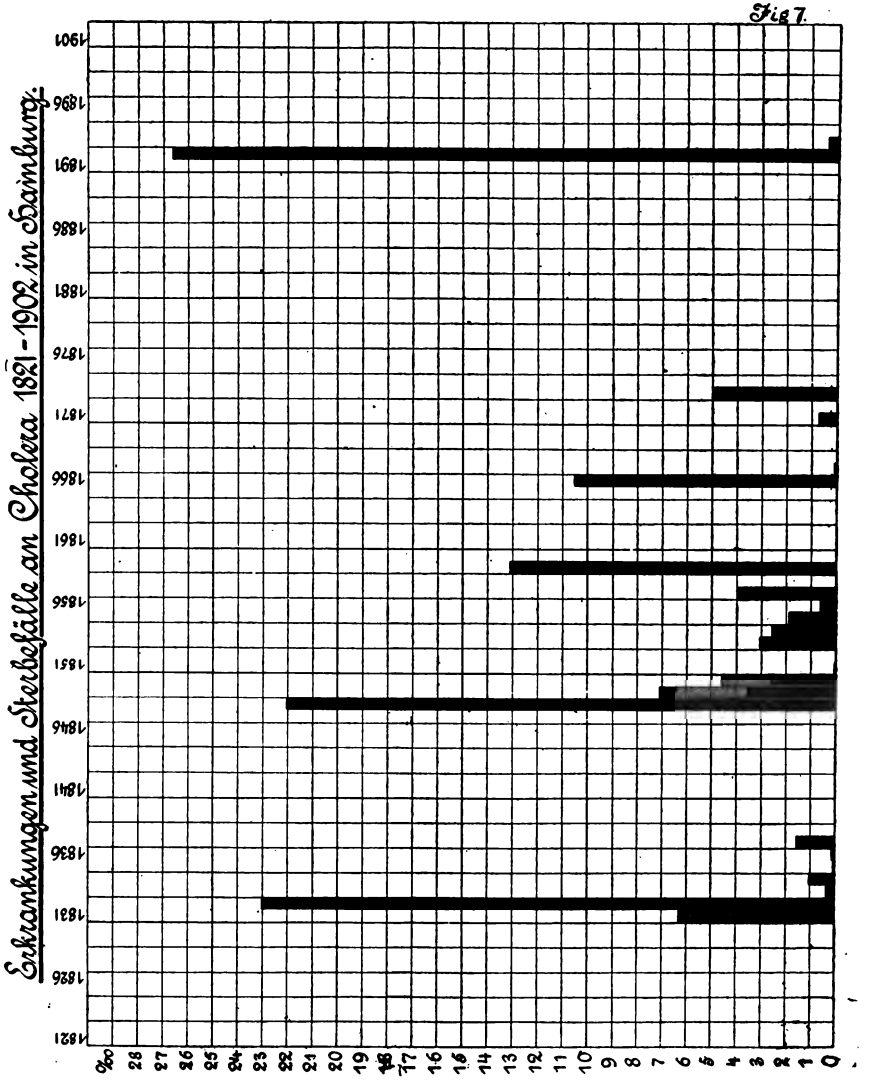
Here I can express myself more shortly, as almost all that I have said about enteric fever can be applied to cholera, which follows the same modes of spread as enteric fever.

On nearly every occasion the disease has come to us by the same classical route that is characteristic for the whole of Germany. It spreads from Asia to Russia, then crosses the German frontier with the floating rafts travelling down the Vistula from Poland. From the Vistula it follows the navigable water-ways of the river through canals into the Oder, and from this river enters the Havel and Spree again by canals, and thence the Elbe. The "Oberländer" ships are always the first with us to be affected by cholera.

It is only in exceptional cases that the route is a different one, as, for instance, was the case in 1892, when Hamburg was attacked before any other place in Germany. The disease then came probably by the sea-route from Paris or Havre, in all probability with some ship's water-ballast taken in at Havre harbour polluted by sewage.

The diagram, Fig. 7, tells you about the outbreak of cholera in our town. The columns show the number of cases per 1,000 inhabitants, the black parts the number of deaths. As you will see, the disease has visited us in Hamburg a good many times. The infection has never come to Germany without visiting us, and remaining with us for a longer or shorter period. This can be easily understood from our relations to water, and from the conditions of our marshes, described above.

The great epidemic of 1848 was not, however, much less than that of 1832. But the sanitation of the town plays as



important a part as it does in the case of enteric fever, and every succeeding epidemic has been less severe than the preceding one. In each of them the scene of action is prin-

cipally on and near the water, or at least among that part of the population connected with the water. From these people the disease is spread further to other portions of the town, that is to say, to parts some distance from the water, lying on the "geest," or high land, and to persons having no connection whatever with the Elbe. Comparatively speaking, Altona always escapes, because its harbour is smaller, and of very much less importance to the town, than that of Hamburg is to the town of Hamburg; and because also the whole of the town of Altona lies high up on the "geest." When cholera does break out there, it is near the Elbe. In the case of these Hamburg epidemics in former years, whether it is a fact that cholera germs also got into the water supply cannot now be determined. It can hardly have been in any considerable degree, on account of the local spread of the infection described above. On the other hand, something of the kind seems to have happened in Altona immediately after the opening of the new central water-supply in 1859. On August 4th of that year the water-service was inaugurated, and it is a fact that at first the filters did not properly perform their functions. Soon afterwards, the largest cholera epidemic Altona has ever known, with eight cases per 1,000 inhabitants, broke out, spreading over the whole town. The information at hand is too scanty to enable us do more than surmise. In Hamburg, where the height of the epidemic was reached in July, there were then 10.5 cases per 1,000 inhabitants. In the epidemic of 1871 matters were already very much clearer. There was at that time a slight outbreak in Hamburg, beginning in August, with 0.5 cases per 1,000 inhabitants. A little later, Altona was attacked. The first cases occurred on August 19th, after unfiltered water had been delivered by the service-pipes from August 11th to 18th. But the number of deaths was three times as great as in Hamburg, namely, 1.43 per cent. as opposed to 0.43 per cent. in Hamburg. The medical authorities of Altona at the very time of the outbreak attributed this epidemic to the water-service, and publicly expressed their opinion.

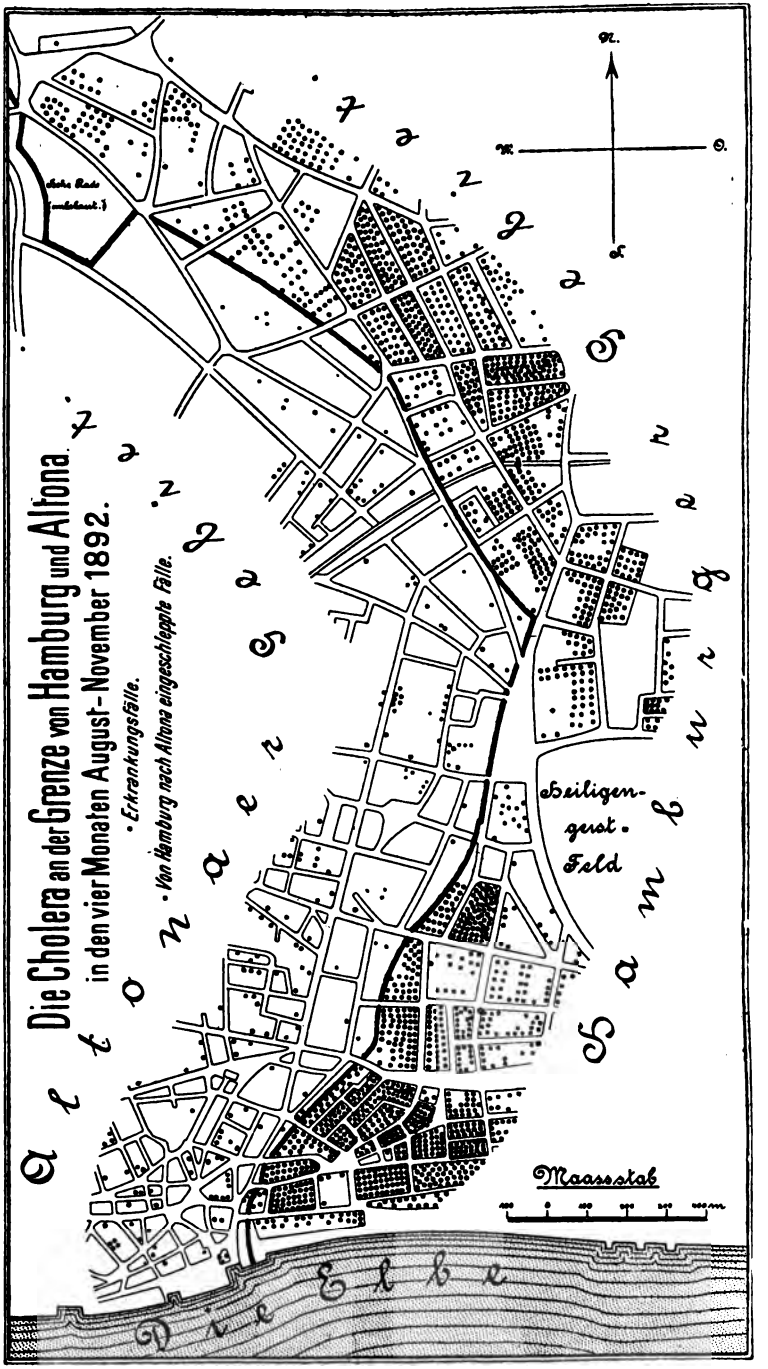
The importance of all these earlier observations on the diffusion of cholera and enteric fever by means of the water-service of the two towns was not thoroughly understood and appreciated till the great cholera epidemic of 1892 broke out in Hamburg, teaching a lesson which could no longer be misunderstood. The essential characters of this epidemic are well known to you, so I can confine myself to following a few points. There were 26.42 cases per 1,000

inhabitants, and 13.44 per 1,000 deaths, whereas in Altona these numbers were 3.81 per 1,000 and 2.13 per 1,000 respectively, and in Wandsbek still less, namely, 3.06 per 1,000 and 2.09 per 1,000. And among the Altona cases were a large number of persons who were infected in Hamburg. The boundaries between these two so widely-differing infected areas follow closely the political boundary, which, as before stated, coincides exactly with the boundary of both public water-supplies. To illustrate this fact, I once more lay before you the well-known plan from Gaffky's description of the epidemic within the borders of Hamburg and Altona, in which plan appear the total number of cholera cases and deaths. On the right, to the east, you will see the streets of Hamburg thickly studded with cholera cases. On the left, to the west of the boundary, you will see the streets of Altona with only very few cases, a large number of which were imported from Hamburg, and which are indicated by a special mark. On the Hamburg side, the whole area as far as the water-service reaches was attacked simultaneously in the same way, whether it was the "geest" or marsh that was concerned, or whether the portions of the town were near to or far from the Elbe. This is a fact which must effectually refute the earlier theory that the marsh was attacked first by the disease, because the subsoil here consists partly of organic materials intermixed with human dejections. The marsh was attacked first in former times only because it lay nearer to the water, and because the water from the polluted marsh-ditches and "Wetterungen" was used for drinking purposes. Now, all at once the "geest," which was scantily supplied with water, and which had hitherto been almost free from the disease, was attacked as severely as the marsh, because, through the water-service, infected water was brought also into these more distant parts of the town, built partly on pure sand, only the year before.

The same circumstances explain how numerous cholera cases imported from Hamburg into the marsh districts near the Elbe gave rise to new small groups of cholera cases; whereas in the "geest" villages, provided they lay far from the river, there was no spread of the disease worth mentioning.

Further, the temporal course of the epidemic is very characteristic of the infection of the water-supply. On August 16th the first cases occurred near the Elbe; on August 21st we had about 100 cases, and on August 27th more than 1,000 a day. No previous epidemic had ever

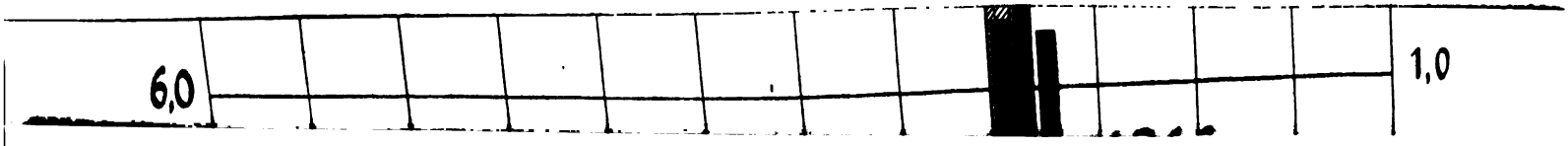
Fig. 8.


Die Cholera an der Grenze von Hamburg und Altona
in den vier Monaten August-November 1892.
• Erkrankungsfälle.
• Von Hamburg nach Altona eingeschleppte Fälle.
Altona
Hamburg
Heiligengeist-Feld
Maassstab
Die Elbe

been of such an explosive character, a fact which is better explained by Fig. 9 than by any verbal description of it. The diagram represents the weekly course of the different big cholera epidemics in Hamburg, not in absolute numbers, but calculated for each week, per 1,000 population, so that the different diagrams can be easily compared with one another. Not even the fairly suddenly arising epidemic of 1848 can be compared with the short, stormy course of that in 1892.

This epidemic, which attacked 16,850 persons and killed 8,576, was followed in 1893 by another smaller outbreak, which could again be traced to the water-service. The water was filtered by this time, but under pressure of circumstance the year before, the work had been hastily brought to a termination, and set in operation. It was therefore possible that unfiltered Elbe water from without entered the conduit conveying the filtered water from the filters to the pump, and mingled with the filtered water. It was not till some days had elapsed that the damage was discovered, and the matter immediately put right. But it had sufficed once more to introduce cholera bacteria into the town, although fortunately only in small numbers. Two hundred and two persons were attacked, and 60 of them died. Since then Hamburg, like the whole of Europe, has been free from cholera. Should the disease reappear, epidemics such as we have had in the past are out of the question, provided very unusual and improbable circumstances in connection with the waterworks do not arise. But after what I have told you about enteric fever, we shall never feel quite secure against cholera. For Hamburg's close connection with the river, the navigation, and the numerous small water-courses, offer enteric fever and cholera only too many opportunities of introducing themselves into the town. We shall, however, be better able to cope with a case of cholera than with isolated cases of enteric fever, because as a rule diagnosis is easier, and the incubation period much shorter, and because it is easier (during the short months of the danger from cholera, which occurs for some months' duration every ten years or less), to direct the attention on this one point, than to carefully observe year by year every isolated case of enteric fever.

That, gentlemen, is roughly the epidemiology of enteric fever and cholera in Hamburg, considering each disease separately. What relation do they bear to one another? Past experience has already taught us that enteric fever and cholera epidemics repeatedly coincide with one another,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which is not to be wondered at in face of the great similarity of their modes of spread. Not only do both seek out similar localities in which insanitary conditions exist in connection with the excrement disposal and the water-supply, not only are both associated in their spread with water, but also dry years and dry seasons with low level of subsoil water are favourable to the development of both diseases. It must, therefore, necessarily follow that when cholera breaks out in a locality, enteric fever, which is usually indigenous there, seizes the same opportunity for further development. In Hamburg it has been repeatedly observed what a peculiar interest attaches to those years in which both diseases were disseminated simultaneously side by side throughout the town by means of the water-supply. At those times enteric fever broke out two to three weeks later than cholera, corresponding to the different lengths of the period of incubation. This was established conclusively in 1892 and 1893.

And a similar connection exists also between these two diseases and choleraic diarrhœa. Choleraic diarrhœa, together with other causes, is brought on after a very short incubation period by drinking water polluted with sewage. In this connection the best-known example is the illness caused by the consumption of oysters which have come from oyster-beds situated in the neighbourhood of town-sewage outfalls. Some persons are attacked by choleraic diarrhœa soon after eating infected oysters, others fall victims to enteric fever a fortnight or three weeks afterwards, and some suffer from both. Dr. Timbrell Bulstrode, in a very carefully-written report, has lately described such occurrences at Winchester and Southampton. We ourselves made a similar observation in 1893. A great shipbuilder's yard supplied its workmen with Elbe water purified in a filter-bed of its own.

At the beginning of August the whole of the filtering material was taken out, cleansed, and put back again. In so doing the filter was rendered very inefficient. A few days later over, one hundred workmen fell ill of choleraic diarrhœa, in a more or less violent form, and after some time in nineteen of the persons pronounced enteric fever developed. In the same way it can be explained how it is that in the more recent great enteric fever and cholera epidemics in Hamburg, which have been more narrowly observed, there has always been a large number of infants who died of choleraic diarrhœa, who certainly suffered neither from cholera nor enteric fever; and that since the

introduction of a central system of filtration of the drinking water in Hamburg, a decrease of 1,000 to 2,000 deaths occurred annually among infants. Although at present we do not know the origin of choleraic diarrhœa, nevertheless, according to these observations, it is certain that in association with the unboiled water used for diluting the milk or for washing out the milk-pans, it gets into the intestine of the child, and here develops its harmful action. We have also some observations from Berlin, from the district of the Stralaus Waterworks, between the years 1886 to 1893, showing that every time the number of germs in the water-supply increased to an abnormal extent, the number of deaths among infants increased in the same proportion.

Thus, gentlemen, in the war we have been waging against cholera and enteric fever, we have, by the introduction of a purer water-supply, at the same time overcome a third enemy, viz., choleraic diarrhœa.

Here, gentlemen, I come to the end of my dissertation, which has left untouched many important aspects in the epidemiology of enteric fever and cholera, and in which I have been obliged to avoid all numerical statistics. However, I hope that the few points I have brought forward have proved of interest to you, and have been welcome as a contribution to the studies to which your Society has dedicated itself.

Discussion on Dr. Reincke's Paper.

The President: We all very much regret that Dr. Reincke has not been able to be present to read his Paper, and to take part in the discussion to which, I am sure, it will give rise. There are a very great number of points which are well worth discussion in the light of our English experience with regard to enteric fever and cholera, and also in the light of the history of enteric fever and cholera in Hamburg and Altona. I shall reserve the few remarks I have to make until a later period of the discussion, which I will ask Mr. Shirley Murphy to open.

Mr. Shirley Murphy: I have listened with great interest to this Paper, because it comes from the pen of my old friend, Dr. Reincke, whose accuracy of observation and industry in recording the facts that come to his knowledge make any contribution of his a matter of great interest to a Society such as this. Hamburg, too, has interest for us,

because it has been the hunting-ground of disease in a very large way; and also because on account of the serious outbreak of cholera which occurred there some years ago, and because of the improvement since made in its water supply. We are able, through Dr. Reincke's Paper, to see the results that have been attained, and now we are told that still further steps are contemplated—viz.: to supply the town with water from deep wells. It will be a matter of profound interest to everybody concerned in public health questions to see what results follow that; and I hope Dr. Reincke will be good enough, when the necessary time has elapsed, to let us hear from him again what those results are.

Coming back to the story Dr. Reincke has told us this evening of the behaviour of cholera and enteric fever, I think it suffices to impress upon us the lessons which we have learnt in this country as to the very large part which water has in the past played in the distribution of diseases like enteric fever and cholera. The interruption of filtration through freezing of the filters is a possibility in this country, although I believe that our extremes of cold are not so marked as they are in Hamburg; but such occurrence shows that filtration is not always to be depended upon; and there is a risk, and always will be a risk, so long as water is derived from a source that is sewage-polluted.

I was particularly interested in that part of his Paper in which Dr. Reincke refers to the behaviour of diarrhœa, and the reduction in the number of deaths from infantile diarrhœa following the improvement in the water supply. Infantile diarrhoea, as we all know, is attributed to many causes; but I do not think that generally the water supply has been credited in this country with largely influencing infantile mortality from that disease.

We shall be glad to learn how far a new water supply will further reduce the amount of typhoid in Hamburg; that the new water supply will probably get rid of the remaining typhoid is a matter that, perhaps, we over here should doubt; because, even admitting that some of the typhoid in Hamburg is due to the water supply, we still have before us in this country evidence that the disease does spread in other ways, and very largely from person to person—a thing which has been recognised for a considerable number of years; and we have also the story that other food supplies have played some part in typhoid prevalence. I may refer to the important work of my colleague, Dr. Hamer, who has very satisfactorily shown,

I think, that the poorer class of fish that comes to London does undoubtedly play a part in this, and he is still working on that subject. Then, again, we have our evidence as to shell-fish and as to water-cress—in fact, there are many opportunities for sewage-polluted food to find their way into our towns. I do not know enough of the circumstances of Hamburg to speak positively, but I can well believe that some remnant of this fever may be due to causes of that sort.

DR. SEATON: I think we shall all agree that this has been an exceptionally interesting and important Paper, especially from the historical point of view. We have the story of the old water-spread epidemics, with which we were unfortunately so well acquainted in this country many years ago. But I agree with a great deal that has fallen from Dr. Murphy, and I think that, in this country at any rate, we must look to other causes as explaining the incidence of typhoid fever. I should say (if I may hazard an expression of opinion) that very little typhoid in this country is caused by water supply, and that, on the contrary, we have to look to other sources. In that connection one is especially interested to note what is said here about the deep well water. Nottingham is a town that has had its water supply from deep wells for many years, and although typhoid has decreased in that town very remarkably during the last thirty years, it is still persistent there. It is one of those towns which has a less favourable record than the Metropolis, whose water supply, as Mr. Shirley Murphy has said, is derived from a sewage-polluted source; so that rather makes us sceptical as to the theory that typhoid is mainly a water-borne disease—in our own country, at any rate.

We still have as many as 4 to 10 cases per annum amongst 10,000 of the inhabitants of this part of the country. No doubt this is too much; but I am afraid a good many of our large towns in the Midlands and the North of England, as well as in Scotland, are worse off than us. I cannot remember what the rate is for the towns of Liverpool and Glasgow—towns supplied with water from the Uplands—but I imagine it is a good deal more than that; at any rate, I know a good many towns where the rate is considerably higher than that, and where the water supply is not suspected of any pollution.

Turning to another aspect of the question raised in this Paper, one could not help being struck by the observation

that is made at the end, about the prevalence of infantile diarrhœa—zymotic enteritis, as we call it—being attributed to water. I think it is many years ago now since we agreed that the prevalence of that disease in Leicester and other towns could not be explained in that way.

I think the record of this Hamburg and Altona experience is one that is of a classical nature, and we value Dr. Reincke's Paper very much indeed for that reason. We are not altogether unacquainted with epidemics in our own country of the same kind: we had the Maidstone and the Worthing epidemics a short time ago. But, on the other hand, we have to remember, in our practical sanitary administrative work, that our chief efforts have to be directed elsewhere to eliminate every possible cause of this still persistent epidemic disease.

DR. NEWSHOLME: The diagrams illustrating Dr. Reincke's Paper are very interesting and instructive; and I hope we shall have an opportunity of seeing more of them, and that they will be reproduced in our *Transactions*. The main diagram reminds me very strongly of a similar diagram shown to the Society by Dr. Childs, when he gave his interesting account of the incidence of typhoid fever in Munich; and one cannot but be struck by the resemblance of the facts in Munich and Hamburg, with regard to the connection between rainfall and typhoid. Although the oscillations of rainfall and fever to some extent coincided also at Munich, yet the connection ceased to be maintained after certain alterations had been carried out to the water works; although, previous to this, both dry seasons and low rainfall had been agents in producing the typhoid, yet they did not produce it in the absence of gross pollution of the water supply.

I quite agree with what speakers have said as to the residuum of fever which still exists in Hamburg. I shall be very much surprised if it does disappear when the new supply from deeper wells is obtained. I think there can be very little doubt, as Dr. Seaton has pointed out, that the greater part of the typhoid fever now prevalent in our better-managed centres of population is not due to water, but is due to other sources of infection—contaminated food—and above all, in a large number of cases to personal infection. I think personal infection operates in a very much larger proportion of cases than we usually give it credit for, and not only in recognised cases, but in unrecognised cases, "carrier cases," and cases of persons who are

not cleanly in their habits. I remember one remarkable instance of a man who was an inveterate oyster-eater, and also a great drunkard. He never got typhoid fever, but he got dead-drunk every night, and had to be put to bed by his housekeeper; and in connection with this drunkenness certain accidents frequently happened, and the result of that was that his housekeeper was infected with typhoid fever. Although it is impossible to prove the connection between this attack of typhoid and the condition of the man, yet I believe the specific poison of typhoid fever passed through his alimentary canal, and thus infected his housekeeper. There are also other instances which have occurred, owing to the chronic nature of the infection in the urine of typhoid-fever patients. I have known several cases of this kind at public-houses. There is at least enough evidence to show that personal infection is much commoner than we give it credit for; and, notwithstanding all precautions, I think there will be a residuum of typhoid fever which will remain until more cleanly habits prevail.

I was struck also with the remarks made in the Paper in regard to infantile diarrhœa; or, rather, as Dr. Reincke calls it, choleraic diarrhœa. The question arises whether we are not speaking of two different diseases. It is quite possible that we are, and that the diarrhœa of which Dr. Reincke speaks is really true choleraic diarrhœa, and not the infantile diarrhœa of which we speak. Otherwise, one cannot understand his associating the diminution of this form of diarrhœa with the purer water supply; because I do not think there can be anything clearer than the conclusion which everyone has arrived at in this country, that infantile diarrhœa has no special relationship, in the vast majority of cases, to the drinking of contaminated water. If there had been any such connection, infantile diarrhœa ought to have diminished in prevalence almost *pari passu* with typhoid fever, which it has not done.

LIEUT.-COL. FIRTH: I doubt, Sir, after what has been said already by previous speakers, whether I can make any useful observations on the Paper which we have just heard. Of course, we as a Society are undoubtedly indebted to Dr. Reincke for his Paper, inasmuch as it constitutes practically a classical summary of the incidence of water epidemics in Hamburg and Altona generally. One point has struck me peculiarly—and in this the author's conclusions are unlike those of workers in the same direction in England—what has struck me very much, has been the fact that he

is still dominated by the idea that water is the chief cause of the dissemination of enterica; and I am pleased to note that such influential workers as Dr. Shirley Murphy and Dr. Seaton have already laid stress on the fact that there are other factors at work. If you take India, for instance, and read India for Hamburg and Altona, we have precisely the same thing going on there. Exactly in proportion as care has been taken to supply good water to communities, so has the cholera incidence fallen; in fact, at the present time, in that country, cholera is the exception rather than the rule, whereas formerly hardly a year went by without severe outbreaks of cholera. But we have not reduced enterica at all—we are suffering from that just as much as in the days before we reformed the water supply. All this points to other factors being at work; and I quite agree with what has been said, that our German colleagues will not find that their deep well, or any other improved water supply in Hamburg, will entirely get rid of enterica.

A question upon which stress has been laid by previous speakers is in regard to the choleraic diarrhœa referred to in this Paper. Everyone seems to have been struck by the remarks of the author. It has occurred to me that Dr. Newsholme is right, and that we are thinking of two different diseases; that Dr. Reincke does not refer to the infective diarrhœa in infants such as we speak of in this country. He really refers to a form of diarrhœa that the Americans have laid great stress upon as occurring in their towns, and in which the causative organism is more or less identical with the *B. dysentericæ* of Shiga; and if this is so, it is quite distinctive from infantile diarrhœa, in which the infective agent is probably a *Streptococcus*. It is not difficult, then, to see the force of his argument, because the dysenteric group of organisms are no doubt sewage organisms, and they are naturally found in sewage, and they are probably to be found in great numbers in the Elbe water; and being there, should there be any excess of this Elbe water in the drinking supply, they would get the organism distributed to consumers. Of course, this is only guess-work, and in the absence of more precise facts it is difficult to do otherwise than guess. But I think we must be careful, whilst giving due appreciation to the facts of this Paper as representing an elaborate summary of German experience, not to lose sight of our own experience; I do not think we are quite justified in arguing entirely from their facts to our own, because in our case the evidence is so strong and so complete that water

is really a subsidiary factor in the dissemination of enterica. We have still much to learn as to the spread of enterica; but, speaking for myself, and in the light of our experiences in the army, I am convinced that it is probably polluted food, rather than polluted water, which is the main cause of the disease. With regard to cholera, it is otherwise. I think there can be no doubt that water is the chief agent of dissemination of that preventible affection.

DR. FRANKLIN PARSONS: I was formerly Medical Officer for a district situated similarly to the district which Dr. Reincke describes, along the head of the Humber, where the water supply was derived from the rivers which had received the sewage of the large towns of Yorkshire; and enteric fever cases were much more frequent in the villages along the course of those rivers where the people drank the river water, than in those inland villages which depended either upon rain-water or upon shallow wells, though these were often polluted. The same thing has been observed in the Trent-side villages in the Gainsborough district in Lincolnshire. Dr. Bruce Low has reported on the villages along the course of that river; and I noticed in a recent report that the proportion of enteric fever cases in those villages where the tidal water of the Trent is drunk, was much larger than in the inland villages. The arrangement of the houses in these cases is not quite the same as in the case of the marshes of the Hamburg district. There is not a ditch running parallel inside the river bank with the houses between; but the people drink the water direct from the river: and they generally take it at low water, because the water is then fresh, whereas at high water it is brackish in the lower reaches.

With regard to enteric fever not being thought to be so frequently associated with water now as was formerly supposed, may not that be due to the fact that so much attention has been given to that particular mode of propagation of enteric fever that there are not now so many polluted water supplies; and that where water is still taken from the rivers it is better filtered, whilst in many cases unpolluted water has been substituted for it?

There are still, however, plenty of cases reported from country places, of outbreaks of enteric fever in connection with polluted water supplies. Not unfrequently such an outbreak is limited to a group of cottages which get their water from a particular well; and sometimes it can be shown that prior to the outbreak the specific infection of

enteric fever has been introduced into the locality, and that it has had opportunity of getting into the well. But even in country places the wells are better looked after now than formerly, and consequently one would expect that the propagation of enteric fever by water supplies would not be so common now as it was formerly considered to be.

Of course, there have been in this country large epidemics of enteric connected with specific contamination of a town's water supply, such as those at Worthing and Maidstone, and Newport, Isle of Wight; and several of these have been preceded by outbreaks of diarrhœa for two or three weeks. Before the occurrence of the enteric fever there has been a prevalence of diarrhœa, and, if I remember rightly, such diarrhœa has not been confined to children, but has attacked older people as well: so that it would look as if that water-borne diarrhœa was not the same disease as the epidemic diarrhœa of children which causes such mortality in hot summers. Also, several outbreaks of diarrhœa attacking people of all ages have been described in connection with contamination of water supply—there was one last summer, described by Dr. Thresh, at Chelmsford.

It seems clear that the ordinary snmmer diarrhœa, so fatal to children, has little connection with pollution of the water supply. During the wet weather of the summer of 1903, the water supplied to London by several of the companies deriving their supply from the river Thames was for a time inefficiently filtered; but nevertheless, in spite of a large number of bacteria in the water, the number of deaths in London from diarrhœa was very considerably below the average all through the early months of the summer, up to the latter part of September. Later on, in October, the diarrhœa deaths somewhat exceeded the average: this excess being coincident with a temperatnre above the mean for the time of year.

DR. E. W. GOODALL: Allusion has been made to the proposal to obtain in future the water supply of Hamburg from wells. A few years ago, a paper was read before this Society by Dr. Saltet, of Amsterdam, in which it was shown that while in Amsterdam, and indeed in Holland generally—a country not unlike that in which Hamburg is situated—there has been a great improvement as regards typhoid fever in consequence of the attention that had been paid to the water supplies, yet that the disease was more prevalent in those towns and communities which

obtained their water from wells than in those which drew it from rivers. So that it does not appear as if Hamburg can expect much more improvement by altering its water supply in the way intended.

An interesting point that has been touched upon is the occurrence of epidemic typhoid fever and cholera at the same time. Dr. Reincke has pointed out that in Hamburg the typhoid-fever rate went up when cholera became epidemic. I looked up the records, as far as I could get them, of a city for which it would be very interesting to have accurate details, namely, St. Petersburg. St. Petersburg is built in very much the same sort of way as Hamburg; the foundations of the house are made by driving piles into the soil. It occupies a site that has been reclaimed from the marshes. It heads the list, I believe, of European cities with respect to typhoid fever, which is very prevalent there. They had cholera rather badly in St. Petersburg shortly after they had it in Hamburg; but when they got cholera they did not have so much typhoid fever. Doubtless the question of diagnosis has to be considered, for perhaps a good many cases of typhoid may have been set down as cholera. Cholera, however, is a more fatal disease than typhoid, and is fatal in a shorter time; so that many persons may have fallen victims to cholera who, had they lived a little longer, might have shown symptoms of typhoid fever; and thus the death-rate from this disease may have been lower than at times when cholera was not prevalent.

From clinical experience I am inclined to believe that there are not a very large number of unrecognised cases of typhoid fever. Indeed, in my opinion the number is over- rather than under- estimated in notifications and death registrations.

We are all extremely indebted to Dr. Reincke for his interesting Paper.

DR. WILLOUGHBY: I did not intend to speak this evening, but I would suggest with regard to Holland, that perhaps the reason why the river drinkers are freer from enteric fever may be that there are so few sewers in Holland. Sewers are almost unknown there, and therefore in drinking from the rivers and canals, the people would not be drinking sewage-polluted water. The whole of the sewage in Holland goes direct on to the fields.

I do not think Dr. Reincke expects to see enteric fever disappear altogether, because he says: "I should like to

remark that several large towns of Germany, supplied with deep well water, have not less enteric fever than Hamburg."

No doubt Dr. Parsons' explanation as to the relative importance of water as a cause is correct, and that it is less prominent now than formerly, because of the improvement in water supply, and the methods by which rivers are freed from sewage-pollution.

As regards Holland, again, the whole of the sewage being applied directly to the land, the shallow wells are much more likely to become polluted than the rivers.

DR. REGINALD FARRAR: I should like to touch on a side-issue of this question, which does not bear directly on the Paper, but I venture to think it has importance in relation to the epidemiology of cholera, and especially to cholera administration in India, where I had experience of a serious epidemic. The incident occurred a few years ago, at the time when Haffkine discovered—or thought he had discovered—that water treated with permanganate of potash killed the cholera vibrio. The Government acted on his suggestion, and issued an order during the famine year 1899-1900, that whenever cholera appeared the wells were to be treated with permanganate of potash, or, as it was popularly called, pinked. The method acted in a very remarkable way on one particular occasion. This being a famine year, and the monsoon having practically failed during 1899, the nullahs in nearly all the camps dried up, and we depended for water supplies on shallow wells which were locally called Shiradis, and which were dug by the side of the nullahs. These were guarded by good-caste water drawers, and they were ordered to serve the people with water in the buckets which we supplied to them. The well was surrounded by a thorn fence, and instead of people being able to dip their own vessels into these wells, they were only allowed to have water poured out by the water-drawers into their vessels. And here I may say that the caste system (which is in its origin a sanitary system) came in very much in aid of modern sanitation: in many ways we found that the caste system enabled us to improve sanitation. But there was one particular camp in my district—it was almost the only camp where the nullah did not run dry; there was still a certain amount of water flowing through the nullah—and the natives, especially low-caste natives, have a great preference for running water if they can get it, and

it was difficult to prevent them from drinking out of this nullah. On one occasion I visited this camp, and the native medical officer told me he had 6 or 8 cases of cholera. Well, I saw those cases isolated in some huts, and I said: "Yes, that is cholera right enough; and there is an order that the wells have got to be pinked." So the wells were treated, just enough permanganate being used to colour them a faint pink; and the consequence of that was that we had an enormous outbreak of cholera, because the people would not touch this water: they would not drink the well water any longer, but they all flew to the water of the nullah. I left that camp, and the next evening an urgent message reached me that one hundred people had died of cholera, and that they were flying in all directions. I went back next day about mid-day, and when I came to the camp and the quarries, I found them all deserted, with nothing but corpses lying about. I rode on a little further, coming upon more and more corpses, until I came to the nullah, in which many were lying, some dead and some dying. I then crossed over the nullah, and came into the camp itself: it was like a battle-field, strewn with corpses in all directions. To put the matter shortly, we had 400 or 500 deaths from cholera. The people scattered in all directions, and spread cholera in neighbouring villages.

I think this is an interesting example of how sanitary legislation may have a totally opposite effect to what it was intended to have; and it also shows how Haffkine's measure is likely in many instances to do harm rather than good. I do not know whether treating the water in this way does or does not kill the cholera vibrio; but, at any rate, its practical effect is to prevent the natives from drinking the water which is so treated, and to drive them to the waters of the streams which are polluted with excreta. I think this is a point of sufficient importance to bring before the Society.

DR. CHRISTOPHER CHILDS: I am reluctant to trouble you with any remarks, because I have not had time to study the Paper, and there are so many details that one can hardly discuss a Paper of this kind straight away without studying it a little. I am pleased to hear we shall have it printed *in extenso*, and also that we shall have these diagrams reproduced, because certainly they are full of instruction.

If I may just make one or two remarks, there are two strong points brought out by the writer. The first is the case for water supply as a vehicle for the spread of cholera.

The diagrams presented by the writer speak eloquently for themselves. The municipal boundary marks the demarcation between the different water supplies, and also between the great majority of the cholera cases on the one side and the merely sporadic cases on the other. I think anyone who opposes the idea that this epidemic was spread by water is at once brought face to face with great difficulty, if he wants to explain how it is that on this side of the line you have all these numerous cases of cholera, whilst at the same time on the other side of the line you get merely sporadic cases.

Then there is the other point of great interest to us, and that is one which has been dealt with—I mean, the effect of sand filtration. We are all familiar with the history of the Hamburg epidemic, and Altona with its much more polluted water supply almost escaping the epidemic at first. Then, there is that sharp outburst in Altona in the following winter, and the discovery that the filters have been frozen; that they have been consequently broken up; and that, instead of about 100, thousands of organisms per cubic centimetre had passed through those filters. We are indebted for this information to Dr. Percy Frankland, who has dealt with this subject in his book on *Micro-Organisms and Water*. And that is brought out again in connection with the typhoid epidemics. Here we see epidemics of typhoid in Hamburg — very extensive, but graduated, always graduated—the curves are not sharp, whereas the Altona outbreaks are explosive outbreaks, and are always in the winter.

Now the question arises—Is this really cause and effect? Are these explosive outbreaks in Altona due to the breakdown of the filters? This appears to be a matter of very great importance, especially to us in London. We depend almost entirely upon sand filtration; and these facts seem to point to the risk that if the sand-filters break down, then we may expect explosive outbreaks of enteric fever. Such explosive outbreaks we do not get in this metropolis, and apparently our sand filtration is fairly effective. Certainly, these points seem to me to be both very strongly illustrated in the history of these epidemics, and in these excellent diagrams.

There is a remarkable correspondence between the typhoid curves shown in these diagrams and in the well-known Munich chart. . . . We have epidemics following the fall of the ground-water as they do in the Munich chart; and it is the more interesting, because in the case of Munich, apparently the meteorological and geological conditions are different. As far as I can understand from the description of the geology of the neighbourhood of Hamburg, you have not there a subterranean river, which is apparently what is the condition in Munich; but you have a more or less permeable soil. In the case of Munich the subsoil water is merely an extension of the river Isar, and the rise and fall of the subsoil water does not correspond with the increase or diminution of the rainfall: it follows generally some period after that. One curious feature about the Munich evidence was this: that when the subsoil water falls the water becomes invariably purer, when it rises it becomes more polluted. I studied the reported chemical analyses of the water supplies in Munich for a large number of years, and worked out the curves relatively to the curves of the epidemic; and the result, generally speaking, was that when the water was purer out broke the epidemic, and when the water was polluted the outbreaks ceased. The explanation of that seemed to be that when the subterranean river rose, it licked up all the impurities in the soil, and became much more polluted. The chemical analyses, at any rate, showed that when the subsoil water rose it became polluted, and when it sank it became purer—that was the curious point. I think we still have a good deal to learn about these conditions of the soil; and all these points are of very great interest, when we compare the results in the various cities.

With regard to this question of the water supply and the outbreaks of cholera, the evidence here differs from the Pettenkofer evidence. Here you have the water supply of Hamburg, there of Altona, and where the water supply is different, there you get this difference in the cholera incidence. Pettenkofer began his inquiry as a firm believer in the English idea that water is the vehicle of of typhoid; but when he had traced out all these cases cholera and their water supplies (inquiring for that purpose into the circumstances of every house in the City), he found there was absolutely no relation between these many different water supplies in Munich and the outbreaks of

cholera. In many cases he would find a certain district supplied in a certain way, but only a certain part of that district attacked with cholera; or where a certain number of streets were supplied from one well, cholera would be only in one of those streets, and would not appear in the others at all. Therefore, his evidence was collected with a bias, but in the opposite direction to the conclusion to which he ultimately came. All these inquiries were recorded, and I had the opportunity of consulting these records. The strong point of Pettenkofer's evidence was really this personal evidence: he tried to trace out the connection of typhoid and cholera with the water supply. It must be remembered, too, that when he supplied the city with pure water from the hills, those who drank it were not relieved from the epidemic.

With regard to epidemics amongst hospitals and barracks in Munich—the barracks of Munich having some 5,000 to 6,000 soldiers—they suffered terribly from the typhoid in the early days; and the curious point was, that these barracks, which were supplied with the purest water, always suffered the most: and so the Munich evidence has always been very disturbing, and shows that there is a great deal yet to be learnt about these things. The result of this is, that although we may accept the general conclusions of Dr. Reincke, we must reserve our judgment for other possibilities. What those are we hardly know at present; but I think we have all lately come to believe that a good deal of this infection may be conveyed either directly or in various ways through the soil.

COLONEL LANE-NOTTER: It is a great satisfaction to hear that the English are coming back to the opinion that there are other sources of infection besides water. I, unfortunately, saw a great deal of enteric fever in South Africa, and I unhesitatingly say that in South Africa enteric fever was, for the most part, spread by personal infection. Over and over again, where camps were drawing from a common water supply, it would appear in one camp and not in another, and we could pick out the tents in those camps which became *foci*, more or less, for enteric fever. And it is easy to see how this was so. If you put fifteen men into a bell tent, where they are in such close proximity, and where the exigencies of service prevent that personal cleanliness which is essential to health: at a time, too, when their powers of resistance is reduced by change

of diet and unusual exertion and labour, and the men are kept up to a certain extent by the excitement of an enemy round about them—all these things contribute to lower the tone of their general health; and the result is that these men are much more susceptible to the attacks of enteric fever—particularly as, under the short-service system, most of our men are from eighteen to twenty-five years of age: an age at which they are more liable to enteric than men who are older. Now, in these circumstances the question of personal infection is a very important one. Not only does it occur through the clothing—and it is impossible when troops are moving rapidly to watch these men closely—it constantly happened that, until they could hold on no longer, and report themselves sick, no one knew there was anything wrong. Then, when a man went into hospital, he would leave his blanket behind him in the tent: a comrade finds it cold, takes it up and uses it, and at once becomes infected. I firmly believe that it was to personal contact and personal infection, and to the common use of latrines, that the majority of cases of enteric fever during the campaign in South Africa were due, and that very few cases, comparatively speaking, were due to water or water supplies. I think that this is an important matter; and I agree with those who have expressed the opinion here to-night that there are other sources of infection besides water in producing these epidemics. One of the principal things we had to deal with was this spread of enteric fever among young men, who, as I have stated, are particularly prone to enteric: the majority of those who went out to the campaign in South Africa were between 20 and 25 or 26 years of age. If we want in the future to effect any improvement in the prevention of these diseases, I am afraid we shall have to look to other things besides mere water supply; and the question is a serious one—how this influence of personal infection is to be prevented. The solution is an exceedingly difficult one. In standing camps, the sanitary organisation of the camp itself may do something and will do something to mend matters; but for the men on the march, exposed to the enemy on all sides, and mixed up as they are with cattle and horses and native followers, it is an extremely difficult problem.

DR. BULSTRODE: Before the discussion ends, I wish to make a proposal to the Society: and that is, that we

should ask the President to kindly convey our best thanks to Dr. Reincke for his most interesting Paper. Papers of this description are those which we should endeavour to get in increasing degree; and I think that we should show our hearty appreciation of them when we are fortunate enough to get then. I therefore beg to propose that our best thanks be conveyed to Dr. Reincke for his Paper.

THE PRESIDENT: I think I may take it that Dr. Bulstrode's proposal is carried with acclamation. I shall have the greatest pleasure, therefore, in conveying it to Dr. Reincke.

Our time is now up. There are a number of other points that might be raised in addition to those which have been already discussed; but I do not propose to do more than mention one or two that have occurred to me.

The first is, that the illustrations that we have had to-night show the great value of diagrammatic as well as statistical treatment of these things. Not only the map to which Dr. Childs has referred—although I agree that that is most striking and valuable—not only that, but the form of the curves, mere annual death-rates, would reveal comparatively little of the real points of difference. The characteristic curves are only obtained by taking into account shorter periods.

The second point is one which Dr. Reincke does not refer to. It would be interesting to know whether in these sudden outbreaks there was or was not a characteristic change in the case-mortality. Personally I should suspect that there was, and I hope to have an opportunity of looking into that question.

Dr. Reincke has given us a history, with notes as interesting and instructive as the history itself, and he indulges in a prophecy. Most of the speakers who have addressed us to-night have ventured to raise a doubt, based on their experience in this country, as to the soundness of the view that typhoid may vanish altogether when a water supply of undoubted purity is secured. Upon that question I should certainly range myself among the doubters. It is perfectly clear that the result of the improvement of the water supply has been an enormous improvement as regards typhoid fever,; but there is a residuum, and the more we inquire into it in all countries, the more we are convinced that there are other causes at

work, which can in many cases be defined. By eliminating the water-borne typhoid which has existed in a community in years gone by, we alter the capacity of that community for typhoid. I suppose it is true that if we have a heavy incidence of a certain disease in any community, and reduce it to a minimum, we thereby increase the susceptibility of the population as a whole, by removing what is practically an acclimatisation.

INDUSTRIAL ANTHRAX—1899-1903.

By T. M. LEGGE, M.D.

(*Read: April 15th*, 1904.)

Any difficulty I have had in preparing this Paper has arisen solely from excess of material. So much has been written on the subject of anthrax, that to have attempted even a summary of this would have left little or no time for reference to the cases which have come within my own knowledge. This is my excuse for making no mention of the work of Dr. J. H. Bell, of Bradford, and of Mr. John Spear, of the Local Government Board on Woolsorters' Disease; or that of the latter's work, and Dr. Hamer's on Anthrax in the Hide and Skin Industry; or of the clinical work of Dr. Davies Colley, of Guy's Hospital.

I ought not, however, to let it pass unrecorded to-night that, twenty-three years ago—on May 4th, 1881—a Paper was read before this Society on "Woolsorters' Disease or Anthrax Fever," by Mr. Spear, which he described as a "subject almost unknown in the annals of British Medical literature." For this reason, naturally, his Paper dealt mainly with the symptoms of the disease. These are now so well known that I have omitted all reference to this aspect, considering that at the present time the epidemiological is the more important one.

I propose to deal with 211 cases of industrial anthrax. They represent practically all the reported cases which have been included in the returns published in the Annual Reports of the Chief Inspector of Factories during the five years 1899 to 1903, both inclusive, as a result of Section 73 of the Factory and Workshop Act, 1901 (previously Section 29 of the Act of 1895). This enactment requires every medical practitioner attending on, or called in to visit, a patient whom he believes to be suffering from anthrax contracted in a factory or workshop, to notify the case forthwith to the Chief Inspector of Factories at the Home Office; and a similar obligation is imposed on the occupier of a factory or workshop, to send written notice of every such case to the certifying surgeon and the Inspector of

Factories for the district. The notification of the practitioner states merely that the case is one of anthrax. The data which form the basis of the present paper were obtained by subsequent inquiry a few days later on the part of the certifying surgeon and the Inspector of Factories. I have selected the period of five years because, during this time all the reports have passed through my hands. In the years prior to 1899, there is little doubt that, owing to ignorance of the requirements of the Section, there was failure on the part of practitioners to notify some cases which occurred. Although, even now, knowledge of the duty of notification is not universal, it can be accepted that, in districts where anthrax is fairly common, information is now obtained of the great majority of the cases. Included in the total 211 are a few cases—not more than half a dozen—in which considerable doubt attaches to the correctness of the diagnosis, but I have thought it well to include them. Taking the figures as a whole, they work out as follows:—

Industry.	Males.	Females.	Total.	Fatal.	Per Cent.
Worsted and Woollen ...	57	15	72	20	27.7
Horsehair and Bristles ...	43	15	58	13	22.4
Hides and Skins	69	—	69	17	24.6
Other Industries	10	2	12	5	41.7
Total	179	32	211	55	26.0

The fatality of these reported cases is considerably less than that usually attributed to the disease, and certainly less than that observed in some early outbreaks. It is, however, almost the same as the following figures, which I have obtained from French, German, and Italian sources. Thus, in St. Denis and the Department of the Seine, between 1886 and 1893, among 123 cases there were 35 deaths—a proportion of 28.4 per cent.* Of 91 cases in the horsehair industry in Germany, 29 proved fatal—a proportion of 31.6 per cent. In Italy† in the eleven years, 1880-1890, there were 34,052 cases, of which 5,812 were fatal = 24.1 per

* *Maladies Professionelles*, Paris, 1903, p. 83.

† *Sullo Stato presente della Sieroterapia Anticarbonchiosa*, by Prof. Achille Sclavo. Turin: 1903 p. 2

cent. In Dr. Russell's* cases, which occurred in 1878 in a horsehair factory in Glasgow, of 9 cases 7 were fatal; and of 88 cases reported by Eppinger† as having occurred among rag-sorters in certain paper factories of Austria, between 1870 and 1886, 78 proved fatal. It is a remarkable fact that the whole of these 88 cases appear to have assumed the form of internal anthrax, whereas of the 211 cases under review not more than 5 are of this type. Even Mr. Spear considered at the time he wrote that the internal form was the more common in Bradford.

The total number of persons employed in wool in the United Kingdom in the year 1901 is stated to have been 259,909 (106,593 males and 153,311 females).‡ Of this large number, however, only 1,171 (1,164 males, 7 females) are classed as being engaged in sorting, and 3,093 (1,882 males, 1,211 females) in combing, the dangerous kinds of wool under the Special Rules for Wool Sorting and Wool Combing, making a total of 4,264 persons incurring special risk.

Taking the total cases in the five years (72), and estimating these on the total number of persons employed, the comparative figure of attack is only 0.0028 per cent. At least 56 of the cases occurred to persons employed in the sorting and combing processes of the scheduled dangerous classes of wool. I have excluded from this number cases of outside infection caused by workers manipulating these wools, and cases among spinners and weavers, even when these occurred in factories where dangerous wools were manipulated, and were possibly the result of infection from insufficient separation of one process from another. On the number (4264), therefore, exposed to risk, taking these 56 cases, the comparative figure of attack becomes appreciable—1.3 per cent.

In horsehair, in all processes, the total number employed in the year 1901 was 2,206 (734 males, 1,482 females). The number of cases of anthrax in horsehair factories was at least 34. I have excluded from this figure altogether the cases which occurred from the use of horsehair and bristles in brush factories. Estimated in the same way as for wool, the figure of risk in the horsehair industry is 1.5 per cent. as compared with 0.0028 per cent. in wool. The risk in horsehair factories generally, therefore, stated

* Supplement of Annual Report of the Local Government Board, p. 321.

† *Die Hadernkrankheit*, Dr. Hans Eppinger, Jena, 1894.

‡ Supplement to the Annual Report of the Chief Inspector of Factories, for 1902, dated 1904.

in this way, shows itself to be greater than in wool, even when the figures dealt with are limited to those manipulating the scheduled dangerous classes. For particular factories, both of wool and horsehair, a very much higher attack rate could be given.

The distribution of the cases according to district is as follows; from which it will be seen that London, and not Bradford, contributes the largest number of cases.

Wool.		Horsehair.		Hides and Skins.		Other Industries.	
Bradford	41	London	23	London	38	Bristol	2
Bingley	7	Glasgow	9	Liverpool	18	Liverpool	1
Kidderminster	11	Sheffield	8	Bristol	3	Halesowen	1
Mirfield	4	Castle Cary	7	Westbury	3	Birkenhead	1
Leeds	2	Worcester	4	Manchester	2	Dewsbury	1
Liverpool	1	Glemsford (Suffolk)	2	Leeds	1	Leeds	1
Sedbergh	1	Salford	1	Keighley	1	London	1
Newchurch	1	Ashton-under-Lyme	1	Tilbury	1	Belfast	1
Edenfield	1	Halifax	1	Penketh	1	Newark	1
Waterfoot	1	Trowbridge	1	Newcastle	1	S. Shields	1
Rawtenstall	1	Walthamstow	1	...		Sheffield	1
Kilmarnock	1	...		...		...	
Total	72	Total	58	Total	69	Total	12

It is, however, necessary to get an idea of the population of the workers throughout the kingdom, to see whether the incidence of anthrax follows closely the lines of relative density of population in the particular industry. This is brought out in the following Table, where for each of the counties in England and Scotland the numbers employed in wool, in horsehair, and in leather, are inserted. The figures for horsehair do not include persons employed in brushmaking. The number of these is 5,365. So far as I have been able to ascertain, however, only 3 or 4 of the cases included under horsehair can be attributed to bristles. The figures as to persons employed are least satisfactory for the hide and skin industry. They include all the tanyards; but it is probable that only very few of the numbers employed in docks, or in wharves and warehouses, among whom incidence of anthrax is greatest, are included.

The following Table shows the number of persons employed in: (1) Wool, worsted and shoddy factories; (2) Horsehair factories; and (3) Leather, in the several counties

of England and Scotland; and the number of cases of Anthrax in the five years, 1899-1903.

[*The figures for* (1) *and* (2) *are those of* 1900, *and for* (3) *of* 1898-1899.]

—	Wool.		Horsehair.		Hides and Skins.	
	No. Employed.	No. of Cases in 5 Years.	No. Employed.	No. of Cases in 5 Years.	No. Employed.	No. of Cases in 5 Years.
ENGLAND.						
Bedford	...	...	...	...	107	—
Buckingham ..	...	...	...	...	28	—
Cambridge ..	...	...	...	...	189	—
Chester	280	...	...		824	—
Cornwall	64	...	...	...	92	—
Cumberland ...	896	...	...	...	390	—
Derby	754	.	...	...	437	—
Devon	1,124	..	...	...	757	—
Dorset	..	...	...	...	35	—
Durham	809	...	...	...	100	—
Essex	35	...		...	123	—
Gloucester ..	3,275	...	...	...	436	3
Hants	...	...	5	...	304	—
Hereford	5	...	...	...	123	—
Hertford	...	...	...	...	169	—
Huntingdon ...	—	—	—	—	—	—
Kent	...	..	...	...	657	—
Lancaster	11,832	5	117	2	3,054	21
Leicester	2,869	...	...	...	214	—
Lincoln	...	...	...	...	425	—
London	227	...	214	24	3,869	39
Middlesex	...	...	18	...	58	—
Monmouth ...	51	...	...	...	20	—
Norfolk	129	...	145	...	83	—
Northampton ...	...	...	...	...	1,061	—
Northumberland ...	34	...	1	.	308	1
Notts	220	...	...	...	775	—
Oxford	945	—	—	—	—	—
Rutland	...	—	—	—	—	—
Salop	585		...	...	155	—
Somerset	1,961	...	121	7	896	—
Stafford	32	...	...	..	898	—
Suffolk ...	...	...	469	2	321	—
Surrey	45	...	...	...	698	—
Sussex	12	...	...	...	126	—
Warwick	1,101	...	...	...	346	—
Westmorland ...	498	...	...	...	46	—
Wilts	2,140	...	...	1	252	3
Worcester	7,263	11	122	4	689	—
York, East Riding	27	...	5	..	852	—
„ North „	4	...	...	...	101	—
„ West „	187.204	55	195	9	4,268	2
Total ...	224,514	71	1,412	49	25,038	69

	Wool.		Horsehair.		Hides and Skins.	
	No. Employed.	No. of Cases in 5 Years.	No. Employed.	No. of Cases in 5 Years.	No. Employen.	No. of Cases in 5 Years.
SCOTLAND.						
Aberdeen	1,245	...	...	...	6	—
Argyll	17	...	...	...	...	—
Ayr	2,766	1	...	..	183	—
Banff	227	...	..	...	...	—
Berwick	299	...	...	...	...	—
Caithness	45	...	...	...	...	—
Clackmannan ...	3,742	...	...	...	68	—
Dumbarton ...	259	...	...	...	3	—
Dumfries	1,032	...	...	...	83	—
Edinburgh ...	1,106	...	...	...	410	—
Eligin, or Moray ...	233	...	..	...	14	—
Fife	215	...	...	...	46	—
Forfar ...	7	...	...	...	221	—
Haddington ...	20	...	...	...	21	—
Inverness	173	...	...	...	...	—
Kincardine . .	42	...	...	...	64	—
Kinross	178	...	...	...	2	—
Kirkudbright ...	857	...	...	...	4	—
Lanark	3,340	...	727	9	531	—
Linlithgow ...	...	...	...	...	28	—
Peebles	2,083	...	...	...	...	—
Perth	774	...	...	..	36	—
Renfrew	1,764	...	67	...	816	—
Ross and Cromarty	29	...	...	...	...	—
Roxburgh	3,240	..	...	...	13	—
Selkirk	4,784	...	...	...	172	—
Stirling	654	...	...	...	...	—
Sutherland ...	23	...	..	...	...	—
Wigtown	122	...	...	...	6	—
Total ...	29,256	1	794	9	15,560	—

It is noticeable that as regards incidence of anthrax on persons employed in wool, the cases are centred almost entirely round the Bradford district of the West Riding, and in Worcestershire. No case at all has occurred in the woollen factories of the Stroud Valley in Gloucestershire, Wiltshire, Somerset, and Devon, representing a population of over 7,000; and again in the whole of Scotland, where nearly 30,000 are employed, only one case has been reported. This is explained by the fact that, practically speaking, except in the two counties named, the nature of the manufacture does not involve the use of dangerous classes of wool in the raw state from Asia Minor and Persia, to be described presently. In the cloth factories of the Stroud Valley and in Scotland, colonial wool chiefly is manipulated. The distribution of cases as regards horse-

hair follows much more closely the population manipulating it, which suggests that the dangerous classes of hair from China, Russia, and Siberia are more generally used. Discrepancies in incidence of anthrax and number of persons employed in leather are to be observed similar to those noted in wool. The counties of Northampton, Somerset, Devon, Surrey, Stafford, Cheshire, West Riding of Yorkshire, and Lanarkshire show practically no cases, although the number of persons employed is fairly large. Anthrax in this industry is mainly confined to the ports of London and Liverpool.

So far as can be learnt, cases in the woollen industry in Germany are exceedingly rare, and I am told the explanation of this is that the dangerous kinds are not manipulated; or, at any rate, only after they have been cleaned and finished in Bradford.

Many factories have had no cases. Incidence is determined by the source of origin of the material, to guard against which, in wool, special rules for the sorting and combing of dangerous kinds apply in 66 factories, and in hides and skins in 118; but even in these the incidence has been on special firms. Cases from wool have occurred in 18 of the 66, and from hides and skins in 44; but in wool 10 cases occurred in one factory, 7 in two, 6 in one, 4 in three, 3 in two, 2 in three, and one case each in the remainder; in hides and skins in one tanyard there were 7 cases, in another 4, in one 3, and in one 2. In one hide and skin warehouse there were 5 cases, in two 4, in three 3. In the horsehair industry, for reasons to be stated later, at present none but voluntary regulations exist.

The Table on page 160 states the precise occupation at which the affected persons were employed. They are given, as nearly as can be done, in the order in which the processes of manufacture are carried on, from the time the material is conveyed to the factory to that when it leaves in a finished condition.

In wool three other cases are included in which there can be no doubt that infection was conveyed to the general outside public by persons working in a factory, and there is one such case also included in horsehair. Anthrax has been apparently contracted from the wearing of a shawl worn previously by another person in a factory, and from the washing of the clothes of a worker.

In wool, distinction must be drawn between the preliminary processes prior to washing and the late combing and carding processes. The remarkable feature of the

Table as regards wool is the large number occurring in the latter processes after it would have been anticipated that the bulk of the danger was removed by sorting, willowing to free from dirt, and washing. Personally I hold the opinion that several of the late process cases are due to infection from the early manipulation, owing to deficient separation of one from the other. But that this will not

Wool.	Horsehair.	Hides and Skins.	Other Industries.
Vanman ... 1	Vanman ... 1	Docks ... 12	Docks 2
Warehouse ... 3	Clerk in Office 1	Wharves or warehouses 34	Fruit warehouse 2
Overlooker ... 1	Warehouse ... 3	Tanneries ... 19	Horn manufacture 2
Occupier ... 1	Opening, weighing and carrying 8	Boot manufacture ... 1	Rag sorting ... 1
Sorting ... 14	Sorting ... 1	Portmanteau making ... 1	Linen weaver ... 1
Willowing ... 11	Willowing ... 6	Leather seller 1	Chemical manure 1
Blending ... 1	Mixing ... 2	Picker strap making ... 1	Saddler ... 1
Washing bowl 5	Drawing ... 8		Unloading corn 1
Mechanic ... 1	Emptying dust-box of willey machine ... 1		Railway station 1
Drying ... 1	Curling and carding ... 8		
Carding and combing ... 15	Brushmaking 11		
Finishing, gilling, and preparing ... 5	Yarn winding 1		
Spinning, twisting, and weaving 4	Stuffing boxing-gloves ... 1		
Waste pulling 1	Mattress stuffing 1		
Dyer 1	Making patent knife-cleaning machine ... 1		
Engine-house 1	Emptying sacks 1		

explain the occurrence of all cases in the carding-room I am satisfied, as is evident from the minute separation of one fibre from another which is effected in combing. If this separation were complete, I believe that cases in the carding-room would occur only occasionally. In the code of Woolsorting Rules issued, no duty was placed on the occupier to remove the dust in willowing; and it is remarkable that in the Report of the Anthrax Committee* no reference to the danger from this machine is made. It was owing to the

* *Conditions of Work in Woolsorting, etc.*, 1897.

occurrence of cases in the carding-room that the later Woolcombing Rules were framed. In 1898, in one factory 8 cases were reported in quick succession, 2 proving fatal. Before the wool was handled by the workers, it was supposed to have been opened, sorted, willowed from dust, washed, dried, carded, and re-washed; but the willowing of the unwashed wool was carried on practically in a corner of the carding-room, inasmuch as in the wooden partition screening it off there were wide gaps and a door. A free passage of air from this willey into the combing-room was assisted by a Blackman fan in the centre of the combing-room. Of the 10 cases which have been mentioned as having incurred in one factory, 8 affected persons employed at combing-machines (no sorting was done here on account of previous incidence on those thus employed); but free communication has existed until recently between the carding-room and the place in which the dangerous wool was blended and washed.

In horsehair, similar distinction is to be made between the treatment of short hair used for curling and stuffing mattresses, and that of long hair to be used for weaving. Practically all the cases have occurred in the processes incidental to curling, because the long hair undergoes a careful process of preliminary washing and heckling. The small number in horsehair due to sorting is remarkable. In the horsehair factory, where 12 cases occurred, none arose in this operation, although as much sorting appeared to be done as would have been the case had the material been wool.

The occurrence of cases in hides and skins in late processes, after immersion of the hides in the liming vats, points to the extraordinary vitality of anthrax spores; although, even in these cases, there is the possibility that infection was conveyed from untreated material.

In the cases included under "other industries" the connection with infected material is clear in those occurring in horn manufacture and in chemical manure works. The saddler more probably contracted the disease from assisting in skinning the carcase of an animal which had died presumably from anthrax than from his occupation. The source of contamination in the fruit warehouse, in the linen-weaver, and in the unloading of corn could not be ascertained. In following up these cases I found that an outbreak of anthrax in horses in London has been definitely traced to grain, and the bacillus isolated from the corn.

Situation of the Pustule.

Situation.	Wool.	Horse-hair.	Hides and Skins.	Other Industries.	Total.
Internal	5	...	...	...	5
Neck	23	26	31	4	84
Angle of jaw	3	1	4	...	8
Upper eyelid	7	2	2	1	12
Lower eyelid	1	1	...	...	2
Cheek	3	11	15	2	31
Forehead	3	5	5	1	14
Scalp	2	1	...	...	3
Face	4	4	...	..	8
Chin	2	1	...	...	3
Nose	1	...	...	...	1
Upper lip	...	...	...	1	1
Lower lip	1	...	1	...	2
Finger	...	...	1	...	1
Wrist	...	...	2	...	2
Forearm	9	3	4	...	16
Elbow	2	1	...	—	—
Upper arm	...	1	...	—	—
Arm and jaw	...	...	1	...	1
Shoulder	1		...	...	1
Chest	...	1	...	...	1
Small of back	1	...	...	...	1
Buttock	1		...	...	1
Sternum	...	...	1	...	1
Foot	1	...	...	...	1
External	2	...	...	..	2
Thigh	...	...	...	1	1
Not stated	...	...	2	1	3
Total ...	72	58	69	12	211

This Table brings out clearly the great frequency of the anthrax pustule to develop on the neck, and its infrequency on the fingers and hand. The case stated to have occurred on the finger is perhaps the most doubtful one included. Recently, however, on a visit to some German factories, I found that the occupier of one of them had lost the two top joints of the index finger of his right hand from what he believed to have been anthrax. I am unable to explain why the finger should not be attacked, because the broken surfaces upon it must afford every opportunity for the lodgment of the virus. The accident of situation is of very considerable moment in determining the severity of the case. Thus, of the 12 cases on the upper eyelid, 5 were fatal—41.6 per cent.; of 84 on the neck 26 were fatal—30.9 per cent.; of 31 on the cheek 3 were fatal—22.5 per cent.; of 14 on the forehead 2 were fatal—14.3 per cent.; and of 23 on the

wrist and forearm only 1 was fatal—4·3 per cent. Great stress, I have since found, is laid on this point by Professor Sclavo in advocating the use of his serum.

Inability very often to venture an operation in malignant pustule on the upper eyelid, and the looseness of the cellular tissue, undoubtedly account for the high fatality in this situation. Occurrence of internal anthrax is limited to wool, and the intestinal form was known to have been secondarily induced in one case in horsehair, where malignant pustules of large size were found in the stomach, and numerous small ones in the intestines; but that no case of internal anthrax has been reported in the hide and skin industry is confirmatory of all that is known of the form assumed by the disease in this industry. Among the 119 cases of anthrax (of which 90 occurred in the hide and skin industry) tabulated by Dr. Hamer,* only one was believed to be of the internal type. The absence of recorded internal anthrax in the hide and skin and horsehair industries cannot be due to failure in diagnosis on the part of practitioners; for of the cases under review those occurring in the hide and skin industry received treatment more frequently in hospital, and in them bacteriological verification was more frequently made than in those occurring in wool. This is brought out in the following two Tables.

The large number in which complete verification of the bacillus by culture, etc., appears to have been made is accounted for by the fact that so many were treated in general hospitals in London, and in towns where bacteriological examination is carried out as a matter of routine. Examination of the reports shows that microscopical examination of the serum alone, or of the blood, is insufficient to base an opinion upon with certainty.

Place of Treatment.

	Wool.	Horse-hair.	Hides and Skins.	Other Industries.	Total.
Hospital	38	40	58	9	145
At home	32	16	8	2	56
Not stated	2	2	3	1	8
Total ...	72	58	69	12	211

* London County Council, Report of the Medical Officer (April 5th, 1894).

Verification.

——	Wool.	Horse-hair.	Hides and Skins.	Other Industries.	Total.
Microscope alone ...	26	1	13	4	44
Microscope, culture, etc.	21	41	43	5	110
Bacilli not found ...	5	2	...	1	8
Clinical appearances only	3	10	2	2	17
Not stated	17	4	11	...	32
Total ..	72	58	69	12	211

And even when bacteriological examination is carried out, failure to find the bacillus is not equivalent to saying that the case is not one of anthrax. Especially is this so when material has to be forwarded to a laboratory at a distance. Failure may be accounted for by (1) the material not being fresh enough; (2) insufficiency of the amount sent; and (3) putrefactive changes in the material during transit. Responsibility, therefore, for the diagnosis of the disease rests much more with the practitioner on clinical grounds than with the bacteriologist. In consequence of the doubt attaching to some cases, and in order to assist practitioners in districts where it might be difficult for them to verify a case, arrangements were made by the Home Office by which material could be sent to a recognised bacteriological laboratory. Dr. Andrewes, in response to this arrangement, has carried out ten examinations, of which in four only was a positive result obtained. As showing the difficulties in the way of bacteriological verification the following case may be cited:—

"A man succumbed to what appeared to be a malignant pustule on the forehead, It was not excised, and subsequent general infection ensued. I was present at the *post-mortem*, and examined blood from the spleen microscopically. Large numbers of what appeared to be anthrax bacilli were observed. Portions of the spleen, lung, pericardial, and peritioneal fluid were despatched the same evening to Dr. Andrewes. The following day he telegraphed that suspicious bacilli were present in the spleen. In his final report, however, he stated that he had been able to find on culture only one single colony of anthrax. Other colonies present consisted of small bacilli

resembling *Bacillus coli communis*; but very few colonies arose in proportion to the amount of material used in making the cultures, the inference being that most of the bacilli present in the spleen were anærobic. The probability is, therefore, that most, if not all, of the bacilli which I saw were of this anærobic kind—*Bacillus cadaveris*"—and had developed *post-mortem*.

In another negative result, where an insufficient amount of material had been sent, he stated:—

"In connection with the negative result in cases of anthrax, I may mention that within the last few weeks I made 3 cultivations from a case of malignant pustule in St. Bartholomew's Hospital, and out of hundreds of colonies of *Staphylococci* and other organisms, I obtained only one single anthrax colony. Had I missed this, the case would have passed as one in which the bacteriological examination was negative."

Professor Trevelyan, of Leeds, reporting on material submitted to him, stated that he found bacilli presenting the characteristics of anthrax in cover-glass preparations made from the specimen, along with great numbers of *cocci*; but that owing to the numerous *cocci* present, it was difficult to get a pure culture of anthrax for several days, as the plates were overgrown with *Staphylococcus aureus*. A mouse inoculated with the broth culture lived, presenting no symptoms. A guinea pig inoculated with a suspension from almost a pure agar culture, died during the night of the second day of the most acute infection, which proved to be typical anthrax.

In a fatal case of typical anthrax, in which, unfortunately, he *post-mortem* was delayed until the fourth day after death, microscopic examination of the blood and spleen-pulp showed, among the vastly more numerous putrefactive organisms of the spleen, some chains of square-cut bacilli, which were probably anthrax; and stained sections of the pustule showed also some bacilli which were possibly anthrax, although they were much obscured by the other organisms with which the tissues teemed. Dr. Andrewes carried out a large number of cultivations in order to try and isolate the anthrax bacillus; and, after a week or more of sub-cultures, at last isolated a bacillus which he at first thought was anthrax. It was pathogenic to mice, slightly pathogenic to guinea pigs; but it proved not to be anthrax bacillus. It was a sporing bacillus, growing like anthrax on agar-agar, but motile, and liquefying gelatine more rapidly. It appeared to him quite identical with an organism which

he had isolated two or three years previously from some Chinese horsehair, and which was described by Bainbridge as *bacillus anthracoides.*

The routine procedure at Guy's Hospital, on a case of supposed anthrax presenting itself, is as follows:—

"In the surgery the bacteriologist at once examines some of the serum microscopically. If bacilli morphologically resembling anthrax are seen, excision is proceeded with at once; if only *Staphylococci* are found, and the constitutional symptoms and appearance of the pustule are not urgent, cultures are made on agar; and as soon as growth of colonies are perceptible (sometimes they can be detected on the same day), the colonies are examined for the typical appearance of anthrax in this medium. But in every case, whether microscopical examination has been considered positive or negative, cultures are always made. Inoculation experiments are not made, as a rule, except in doubtful cases, and then from cultures and not from the original material. In all cases also the pustule, when excised, is examined microscopically. At St. Bartholomew's Hospital greater importance would appear to be attached to inoculation directly from the material, as being the most likely means of giving a positive result. In all the recent cases in Glasgow, verification was made by Dr. Buchanan, Bacteriologist to the City Corporation; in Sheffield, by the Medical Officer of Health; in Worcester, by Mr. Duncan, the County Council Analyst; in Leeds, by Professor Trevelyan; and in Kidderminster, by arrangement with the Clinical Research Association. In Bradford, Dr. Eurich has been appointed by the City Corporation to assist the Medical Officer of Health by bacteriological diagnosis of anthrax; and cases here and in the neighbourhood have been examined, some by Dr. Andrewes, and others by Professor Trevelyan, and others again by Dr. Gammidge. So far as my information enables me to judge, however, verification of Bradford cases in the past has mostly been limited to microscopical examination.

The use that has been made of the arrangement of the Home Office for verification of doubtful cases has not been large, the reasons being probably that: (1) The number of reported cases is comparatively few, and of those that are reported only a very small number are doubtful; (2) the trouble entailed by the conditions imposed in sending the material (registered packet, etc.), is too great for a busy practitioner to attend to; (3) the practitioners in country districts, where it was hoped that the arrangement might

be of use, do not on principle excise the pustule, and, therefore, confirmation or the reverse of their diagnosis, would not in any way affect treatment; and (4) owing to the sporadic nature of the outbreaks, it was impossible to apprise all medical practitioners who might come across cases of the facilities for assisting in the diagnosis. To widen our knowledge of the bacteriology of the disease, it would have been useful also to have obtained copies of reports on every case, or supposed case, of industrial anthrax: the examination into which had been carried a stage further than the mere microscopical examination of the exudation, or pustule.

Treatment.—Of the 211 cases, 145 received treatment at some stage of the disease in hospital, and 58 appear to have been treated entirely in their own homes. In 8, information on the point is lacking. Excision of the pustule was practised, apparently, in 143 cases; in at least 22 instances no operation was attempted, owing, in some cases, to failure to diagnose the disease; in others, to the verification having been made too late; and in others, again, to the situation of the pustule on the eyelid. Caustic only was applied to the pustule in 9; injection of carbolic acid was made in 2; and in 35, information as to precise treatment is lacking. The 9 cases in which treatment was by application of caustic are of interest. None of them proved fatal. All occurred in factories in small villages, where it is possible the physique of the persons attacked was so much better than that of workers in towns like Bradford and London, as to enable them the more effectively to resist the disease.

Interest would attach to detailed analysis of the fatal cases, on the point as to how far delay in seeking operative treatment can be held to have accelerated a fatal issue. Of the 20 fatal cases in wool, 9 died in general hospitals and 10 at home; in one, information on the point is wanting. In 8 of the cases operative treatment was—or would have been, if tried—too late; in 3, the situation of the pustule on the upper eyelid precluded excision; and 4 were of the internal type.

It does not appear that, in this country, treatment of anthrax in the human subject, by means of a serum, has been attempted. In Italy, however, the serum of Dr. Sclavo, Professor of Hygiene at the University of Siena, appears to be now frequently used. In his paper he gives details of 164 cases treated by means of his serum, and in these the fatality was only 6.09 per cent. One case of malignant

pustule in a woman, so advanced that the bacilli were even found in the blood, recovered. He states that, so far as experimental evidence goes, no treatment for this disease has been attended with so much success as serumtherapy. Where death does occur, it is in persons who are already *in extremis* when the injection was made. The next Table gives, so far as this can be done, the source of the suspected material.

Wool.		Horsehair.		Hides.	
Persian	22	China	21	China	16
Persian and Camel-hair	7	Russian, Siberian, and China	7	China, East Indian, etc.	3
Bagdad locks ...	4	South American and Siberian	7	Madagascar, East Indian, China ...	1
Persian, Alpaca ...	1	Australian, South American, and Siberian	1	Bombay	12
Mohair, Persian ...	1	Native American, Siberian and Russian	5	Bombay, East Indian	1
Mohair	11	Siberian	1	East Indian ...	6
Van mohair ...	6	Russian	1	East Indian or Russian green hides ...	1
Mohair, Cashmere, Came-lhair ...	1	Foreign	1	Cawnpore goat skins (doubtful case) ...	1
Cape mohair, Turkey mohair	2	All kinds	2	Cape and Singapore	1
Mohair, Alpaca ...	1	All kinds except Chinese	4	Cape	1
Pelitan, mohair ...	1	Australian	1	Australia (dry) ...	1
East Indian... ...	6	Native	2	South American, New Zealand ...	1
Native and East Indian	1	Native and United States	1	Arabian	1
Camel-hair	3	Indefinite	4	Morocco	1
Indefinite	5	Total ...	58	West African ...	1
Total ...	72			Buffalo	1
				Madras and Bombay, tanned hides ...	1
				Tanned sheep skins	1
				Tanned boot leather	1
				Native	1
				Indefinite	16
				Total ...	69

Roughly, I think that in wool 34 of the 72 cases may fairly be attributed to so-called Persian, and 22 to Turkey Mohair or Van Mohair; whilst suspicion only in a very few cases attaches to Alpaca, native, and least of all to Australian wool. In horsehair, 28 can almost with certainty be ascribed to Chinese hair, and 22 to Russian or Siberian; while suspicion in only a few attaches to South American, Australian, and native hair. In hides and skins, 20 can be attributed to China, 21 to Bombay and East Indian; while native and colonial (excluding East Indian) are exempt. Three were traced to tanned leather.

A mere statement of the source and origin of the cases during 5 years conveys little information that is surprising, unless this is interpreted in the light of the kind of material imported from the countries concerned.

The diagrams appended show the amount of wool, horsehair, and hides and skins imported in 1902, in pounds sterling, from every country exporting more than an amount equivalent in value to £10,000. It is apparent at once from the Table for wool that it is an infinitesimal quantity of the whole which produces the great bulk of the cases of anthrax. The diagrams want some explanation. They relate solely to imports, and, as regards wool at any rate, one-half is re-exported. Thus, out of 743,046,104 lbs. imported in 1892, 430,828,993 lbs. were re-exported. It would be impossible for me to prepare a Table representing the amount of wool from each country consumed only in Great Britain. The figures give the amount exported from the various ports; and in the case of wool, horsehair, and hides, there can be no doubt that the considerable amounts set down as coming from France, Belgium, Holland and Germany include material which, so far as the country of origin is concerned, ought to be added to that from Turkey, Persia, etc. But I have prepared the diagrams in this way. solely to try and obtain a comparative figure by which to estimate the relative danger of the wool, horsehair, and hides from different countries. It must be borne in mind, also, that while I have taken the value of the wool imported in one year only, the cases of anthrax are the figures for five years. The gross figure for wool, as I have given them (including alpaca, mohair, and camel-hair), amounts to a total of £22,198,413; and the comparative figure, therefore, expressed as a value of the material imported for each one of the 72 cases, is £308,311. Taking into account only the 56 cases which can justly be attributed to manipulation of Turkey or Persian wool, and estimating them on the value of the wool and mohair imported from those countries (£967,348), the comparative figure falls to £15,488. Proceeding in the same way for the 58 cases in horsehair and bristles, and estimating them on the total amount of horsehair and bristles imported (£1,098,937), the comparative figure for each case is £18,947. Of these cases at least 20 can fairly be attributed to the manipulation of horsehair from China (£40,758), and for each one of them, therefore, the comparative figure is £2,038. Omitting 4 cases due to prepared leather, the comparative figure for 64

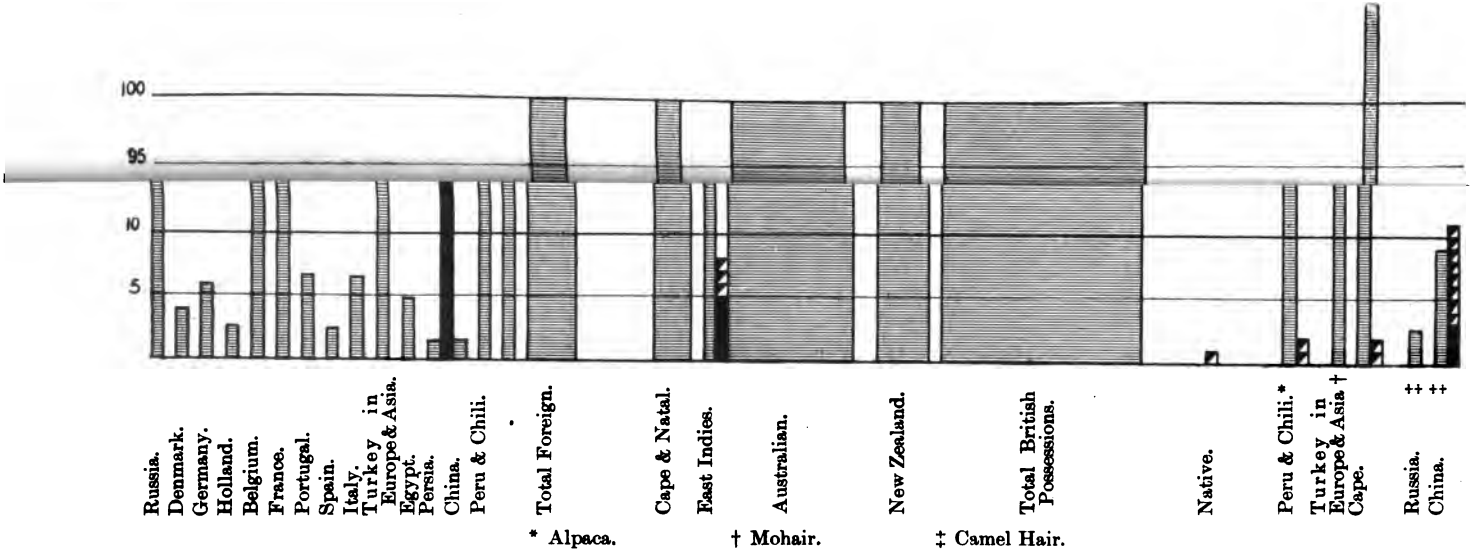
of the cases in hides and skins which can be pretty certainly assigned to the manipulation of dry hides and skins, works out at £46,666. Considering only the 20 cases attributable to China hides or skins (of which the imported value is given as £87,989), the comparative figure is only £4,399.

The figures thus presented are of value in showing that risk of anthrax is greatest in the horsehair industry, and that the risk, both in horsehair and in hides and skins, is very much greater in dealing with material from China than from other countries. The risk from manipulation of Chinese bristles—the value of the imports of which is put down as £147,892—would appear to be vastly less than that from manipulation of horsehair, two cases only having been traced to their use. This is explained by the treatment which the bristles undergo, to be described presently. From wet hides, of which the imported value was £1,595,109, risk of anthrax appears negligeable, as one case only was suspected to have arisen from their manipulation.

The explanation of the greater incidence of anthrax from one class of material over another is to be found, probably, firstly in the extent of prevalence of the disease in one or another country, and the steps which are taken to combat it; and secondly, the extent to which the material is cleaned and prepared before it leaves the country. Less risk is to be anticipated where animals are killed in some central station, as is the case in Chicago and Buenos Ayres, than where the material is collected from widely separated districts. The more uncivilised, from our western standpoint, a country is, the less chance is there of the exclusion of material from dead animals, or of the adoption of precautionary measures. It is held in Bradford that the greasiness of Australian wool prevents the diffusion of dust, and that this is the reason why anthrax is practically unknown from manipulation of Australian wool. On the other hand, I should attribute it to the fact that it is a civilised country. Much Persian wool comes over in a dirty and unwashed condition, and those manufacturers who have thought on the subject at all, have considered that the special incidence of anthrax, from so-called Persian wool, is due to the heat generated by the fermentative processes which must constantly be going on inside the press-packed bale; and they point to the fact that as soon as the iron bands are sundered the band may double in size, owing to liberation of gas.

The question would be solved had we definite knowledge

CHART I.



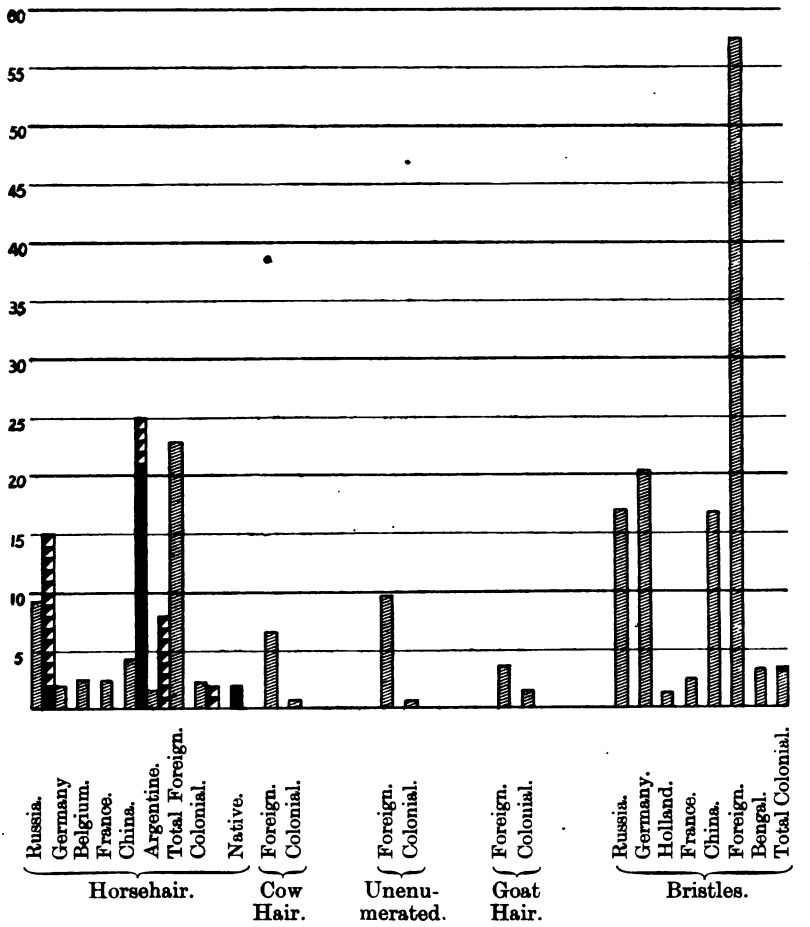
Imports of Wool into United Kingdom in 1902.

(Each division represents value equal to £10,000.)

Each division represents a case.

Suspicion.

CHART II.

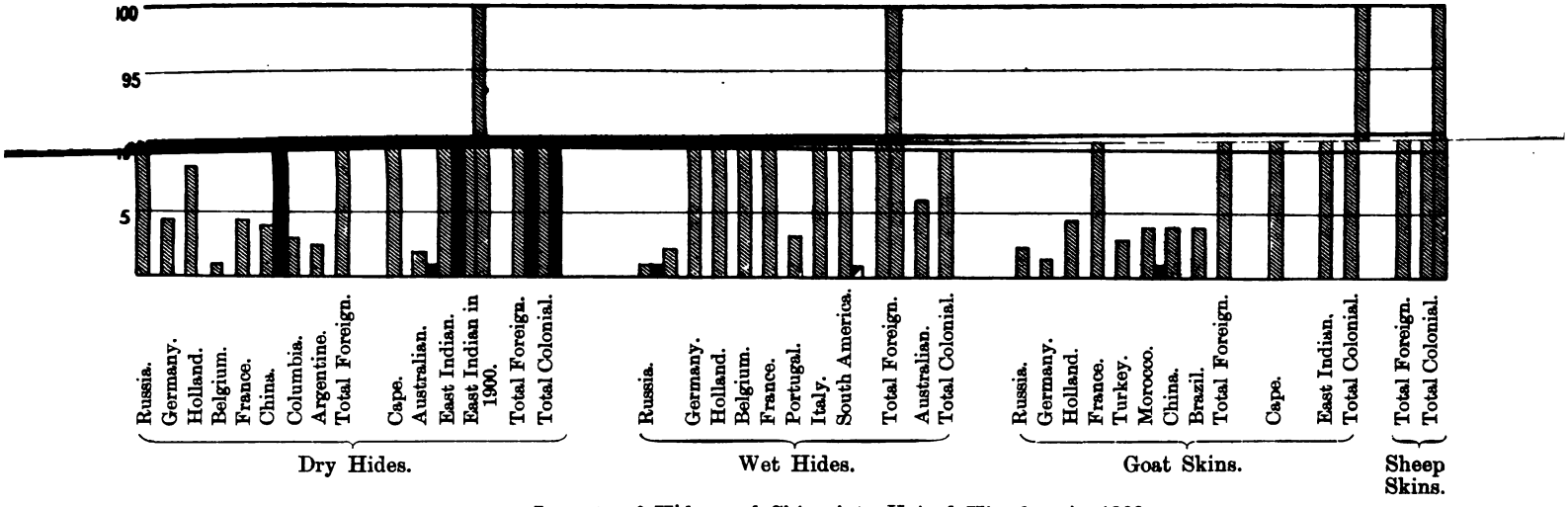


Imports of Hair and Bristles in 1902.

(Each division represents value equal to £10,000.)

Each division represents a case. Suspicion.

CHART III.



Imports of Hides and Skins into United Kingdom in 1902.

(Each division represents value equal to £10,000.)

Each division represents a case. Suspicion.

of the incidence of anthrax on men and animals in Asia Minor, Persia, Siberia, and China; but it is just here that information is scantiest. In conversation with a gentleman who was agent for a Bradford woollen firm for ten years at Stamboul, he told me that in his workshop he employed 30 men separating inferior portions of wool from the rest, and making them up into separate bundles. This was done without assistance from any down-draught, and the dust was shaken out while the workers smoked cigarettes and drank coffee. Nothing was heard about anthrax. Sometimes, on noticing that a worker was absent, he would ask, "Where is So-and-so?" and the reply would be, "Oh, he is ill." A few days later he would ask, "Is not So-and-so better again?" and the reply might be, "He is dead." "What did he die of?" to which the reply was, "Oh, he was ill." He had, however, heard of what was called "shepherd's boil," and he had seen the scar on the neck of one who had had such a boil seared out with a red-hot iron. Disease in the winter among the animals he knew was frequent. Those engaged in the collection and treatment of wool in Asia Minor can tell at once when there is a diseased skin by the odour; and although this gentleman would have had such diseased skins burnt, it would by no means be a general practice.

A report obtained for me by Dr. Clemow of the outbreaks (of animals) in Turkish territory during 1902 mentions only a few outbreaks; but Dr. Nicolle, of the Pasteur Institute, who was for a time Bacteriologist to the Government in Constantinople (to whom I was referred by Dr. Clemow), has informed me that he had been able to satisfy himself frequently of the excessive prevalence of malignant pustule: a fact which he says is not surprising, as in not a few places anthrax destroys at least 30 per cent. of the flocks.

What are the conditions under which the wool is collected in Asia Minor and Persia? And here let me emphasise the fact that a large proportion of what is, in this country, called Persian wool is grown really in Asia Minor, in the districts round Bagdad, and on the borders of Persia. The only report on the subject I have been able to extract information from is one by Mr. S. P. Sweeney, on sheep and wool in Asiatic Turkey, published in 1891.* He says:—"The quantity produced in every district seems to depend on the habits, character, and civilisation of the

* *Wool and Manufacture of Wool*, by Worthington C. Ford, Chief of Bureau of Statistics, Treasury Department, Washington, 1894.

inhabitants of the respective provinces. Thus, the greatest exportation is made from Mesopotamia, where the inhabitants are all nomads, and have no other occupation than taking care of very numerous flocks of sheep, and migrating during the whole year, from one region to another, according to the climate and foraging conditions." In speaking of wool classification, he touches on the condition which must be most likely to cause extensive loss from anthrax once a case occurs, namely, the overcrowding of the flocks in shelters during winter:—"The wool of the flocks of sheep of the plains of Mesopotamia, and which pass the winter outside, owing to the mild climate, is of much finer and cleaner quality than the wool of the flocks of the north, viz., of the province of Koordistan, where the severe winter obliges shepherds to keep their sheep under peculiar roofs always inadequate to cover the numerous animals, which very often lie in dirt."

Of the vilayet of Aleppo, he remarks, that in the colder districts the Bedouins keep the sheep during the winter in caves; and in the district of Diarbekir, that the loss of sheep and lambs by exposure or neglect is small; but in times when disease prevails, the losses are severe. The presence of small rivers increases the chance of the wool being clean and free from dust. In the vilayet of Angora washing is practised, he says, comparatively only on a very small scale, because it does not pay the exporters.

The explanation, apparently, why Persian wool is so largely in demand, is that the breed of sheep is one of the few in which a large proportion of the animals are naturally of a black, brown, or fawn colour. Thus, in the districts around Bassorah, Bagdad and Mosul, the principal family of sheep is of the Chipal breed, of which only about 20 per cent. of the flocks contain white sheep, the remainder being black, grey, fawn, and brown; and the wool is of a finer quality than that of other kinds exported from Mesopotamia. So long as the demand for cloth of undyed brown, fawn, or khaki colour exists, it can at present only be supplied from this so-called Persian wool. The only permanent means, therefore, that I can see, by which incidence of anthrax from wool of this colour can be lessened, is by transporting some of the breed to a civilised country like Australia, and seeing how far there the breed can be acclimatised.

Chinese wool comes from Mongolia. It is said that dirt and sand are wilfully mixed into the strands; and although foreign merchants at Tientsin deduct for all the

dirt thrown out by the cleansing machines, still the practice continues. The percentage of loss from dirt alone is from 10 to 40, and at times 50, per cent. Before being press-packed, it is cleaned as much as possible in machines. In the remote west, the first hands who collect wool have not yet found it worth while to separate the different sorts.

Knowledge as to the manner in which horsehair and bristles and hides and skins are collected and prepared in China is difficult to get at; and similarly, too, knowledge of the incidence of anthrax on animals, and on the persons who handle the material. I have been fortunate in obtaining very interesting information on some of these points from an Englishman in China, the agent of a large London firm of horsehair merchants. The firm readily acceded to my request that he should be asked to furnish what information he could as to the collection and transmission of material in China before it was exported. Hides and goat skins are brought to the markets in bundles, and sold on gross weight, which of course includes the baling, rope, fat, mud and dirt. If the demand is great, anything that has the appearance of a hide or skin is included. If the market is poor, the bundles contain good skins, as the interior Chinese purchaser knows that the market, being poor, the foreign buyer will be particular, and refuse skins which are not good; but these diseased skins, although refused in the interior, are not thrown away. They are stored up until the market improves, when they are placed in sound bundles and forwarded.

In the interior, Chinese agents buy from the Chinese, no foreigners dealing directly with the seller in the interior. These Chinese agents then send sample bundles to the agents of the foreign firms. Fifty per cent. of the horsehair exported comes from Koolan, in Mongolia, on the borders of Russia. Here there are thousands of wild horses. The hair is washed, beaten and bundled, packed in bales, and forwarded by Mongolians or Chinese to Chinese agents in Tientsin for sale. No doubt some hair in this district comes from farmed horses, but all parcels contain dead hair. Another 50 per cent. comes from Mukden, and is the hair from living horses or ponies, which is cut off, washed and bundled. No precautions are taken against disease. The risk from tail hair is small, as it is washed once or twice by the Chinese in the interior, and good material always realises a good price. The great danger is in the manes and combings, which are packed in

bales at Tientsin. Combings, as the name implies, contains the refuse of manes, tails, and includes even hock hair. It gathers all the dirt, and does not pay for cleaning. Hair, like hides, is sold by gross weight. Bristles come to the market in the raw state, and are bought on measurement. The pig is killed, and immediately placed in boiling water; the bristles are scraped off and collected, washed in hot water, exposed to the air and dried, then roughly bundled to measurement, and packed into baskets for sale. These baskets are sold to the interior buyer, who opens, cleans, and re-sorts the bristles, and sells them to the foreigner, who packs them for shipment. The bristles are washed (after having been scraped off the skin) once in very hot water, the hair and fat are removed, but the bristles gather dust and dirt in the drying. No disinfectant of any kind is used. The bristles pass through about ten hands before reaching the foreigner for export. My correspondent adds: "You may rest assured, if the animal had fifty anthrax boils and a good crop of bristles, those bristles would be collected and sold to somebody."

He says that in eight years' experience he has only known of one real case of anthrax in a Chinaman ending in recovery. His own coolies have told him that they were often attacked by boils or swellings after sorting horsehair. They usually went immediately to a Chinese doctor, who cured them by using hot water and a poultice of herbs. Inquiry made by him of Dr. Smith, of the London Missionary Society, did not elicit more than that, in his fifteen years' practice among the Chinese, he had had hundreds of cases of boils, none of which were very serious or ended fatally.

The information he gives as to incidence of anthrax in animals amounts, practically, to the statement that it is "almost unknown."

I do not propose here to discuss in detail the measures of precaution which are adopted to guard against anthrax in the industries in which it may arise, except in the case of horsehair. As there is no means practicable for the destruction of anthrax spores of general application, except steam disinfection, it would appear that horsehair alone can be expected to stand this treatment without being damaged for manufacturing purposes. The generally-accepted method of steam disinfection in this country, with a pressure of at least ten pounds, affects seriously the nature of wool. Hence, remedial measures in this industry are directed towards keeping the anthrax spores below or

away from the faces of the workers during the processes prior to washing, by means of exhaust ventilation. This method is only partially successful, and by it the bacillus is not destroyed but only shifted, and may find its way into water-courses, or it may be used as manure. In Germany, in all horsehair and bristle factories, disinfection of the raw material by current steam under very low pressure has been required since 1899. One manufacturer, Mr. Webb, of Worcester, in this country has, since 1897, passed what is considered the dangerous material (Chinese, Siberian and Russian hair) through a Washington-Lyons apparatus. The experiments on which the German regulations are based were made by Doctors Kubler and Musehold in the Imperial Health Office, and appear to have extended from about the year 1894 to 1900.*

On the whole the German manufacturers appear to have supported the Government; but, owing to objections raised at one time and another as to the inferior quality of material that had been disinfected, according to the Regulations, experimental investigation has been very thorough. The question now appears to have been definitely settled that no microscopical change occurs in horsehair after subjection to current steam, at a pressure of 2¼ lbs. for half an hour, and subsequent manipulation in the ordinary way to set the curl, which can be referred to any injurious action excited by the steam. Experiments were carried out on manufacturing premises, and samples of the hair experimented with were followed through all their later stages of manufacture. The following method was adopted:—1. Horsehair disinfected in the raw state in accordance with the Regulations, and, after spinning, subjected to steam of at least 7 lbs. pressure, to set the curl—*i.e.*, disinfected, steam-curled hair. 2. Horsehair which without previous disinfection was spun, and subsequently steam-curled, as in (1)—*i e.*, not disinfected, steam-curled hair. 3. Horsehair which, without previous disinfection, was spun, and the curl set by immersion in hot water (80 deg. C.)—*i.e.*, not disinfected and curled with hot water. 4. Horsehair disinfected in the raw state, and subsequently treated like (3)—*i.e.*, disinfected and curled with hot water.

These were then subjected to the following tests:—1. Comparison of the loss of weight sustained. 2. Judgment

* *Arbeiten aus dem Kais. Gesundheitsamte*, vol. xv, pp. 456-499; vol. xviii, p. 1.

of the quality by:—(a) Determination of the elasticity of the hair and of the load necessary to rupture it, by means of special apparatus whereby the two points could be determined by readings on a scale; (b) the opinion of experts—the occupier and technical adviser of two horse-hair factories.

Loss in weight.—The conclusions as to this are:—(1) That a properly cleansed sample of horsehair, which has undergone disinfection according to the Regulations, does not lose weight in appreciable amount during the subsequent treatment to convert it into curled hair; (2) with dirty material there is a loss in weight of the disinfected material as compared with that which has not been disinfected, due to the preliminary disinfection causing a greater separation of dirt during the subsequent manipulation. This loss in weight amounted to 11.4 per cent. in the disinfected material, as compared with 10.9 in the undisinfected, a difference of only 0.5 per cent.

Effect on the quality of the hair.—Long Russian tail hair (30 cm. in length) and American mane and tail hair, both in a disinfected and undisinfected condition, showed difference in elasticity so slight as not to count. The conclusion can be drawn, therefore, that disinfected curled hair which has been subjected to steam at a pressure of 14 lbs. to set the curl, when compared with hair treated in the same way without the preliminary disinfection, cannot be regarded as of diminished value or a source of loss.

These experiments, however, gave the key to the divergent views held by the manufacturers and the results of the experiments of the Health Office, by showing that to obtain success, and to preserve the material from damage, disinfection must be carried out within certain definite pressures. The steam-disinfecting machine must be of proper construction, provided with reliable means of ascertaining and controlling the pressure of steam and temperature, and a person properly informed as to its use must have charge of it The following are the points on which stress in construction and use of the apparatus is laid. The steam should enter from above and not from below, for the reason that hair lying immediately over the point at which the steam enters is subjected to a much higher temperature than that registered for the pressure in the centre of the apparatus, because the incoming steam on leaving the boiler is under a pressure of 42 lbs. or more. Further, steam entering from above has the advantage that, steam being lighter than air, it drives the cold air out of

the disinfecting space, and causes all the articles to be disinfected evenly. Condensation is thereby reduced to a minimum, and the chances of wetting the hair is lessened. 2. The pressure guages should be exact, and should have marked on them the particular pressure of 2¼ lbs., which should not be exceeded in the preliminary disinfecting process, and the pressure (7 lbs.) most suitable for setting the curl. 3. Thermometer and manometer should control one another. The thermometer should be placed so as to record accurately the temperature in the interior of the apparatus. 4. Constant attention to the apparatus during the process of disinfection. 5. Protection by non-conducting material of the iron casing of the apparatus, and protection against the weather. 6. It is necessary that press-packed bales should be opened out, as steam will not penetrate so as to disinfect thoroughly, unless the material is loose. In Germany, the disinfecting apparatus generally used is a low-pressure one, and it is probable from the use of high-pressure apparatus in this country that the belief still prevails that horsehair cannot stand steam disinfection without injury. The experiments of Cobbett and Dalton* show that saturated steam, at 100 deg. C., is quite as effective as superheated steam, both in penetration and sterilising effect.

Mr. Webb, horsehair manufacturer, of Worcester, has carried out a number of experiments with Mr. Duncan, the County Analyst, on the disinfection of horsehair in a Lyons Disinfector. His experiments have the additional interest that they have all been carried out with known infected material, and not with anthrax spores artificially introduced into the material. They are of opinion, rightly, no doubt, that these spores are the more resistant surrounded as they are by dirt and grease. They found that material subjected to a temperature of about 245 deg. Fahr. for about twenty minutes appeared to be sterilised, but subsequently anthrax was cultivated from the dust obtained after carding. The carding process, they believe, breaks up the protecting grease and hair, and liberates anthrax spores which have survived the action of the steam. Since then, Mr. Webb has cut open all bundles and bunches, so as to loosen the condition of the hair before submitting it to disinfection. They have found the temperature of 226 to 230 deg. Fahr. as effective as the higher temperature of 245 deg. Fahr. Still they have occasionally found, con-

* *British Medical Journal*, 1900.

trary to the experiments made in Germany, in dust from subsequent cardings of this disinfected material, living anthrax spores, although in a condition of lowered vitality. Mr. Webb states his opinion, however, that "No manufacturer should handle Chinese or Russian mane hair, cow hair, or combings, or any other Oriental hair, without some disinfection at a temperature of 226 to 230 deg., Fahr., for 20 minutes, and that the same process is quite advisable for long hair." Boiling the hair they hold to be distinctly less effective than steam, although boiling the hair for two hours is allowed in the German Regulations as an alternative to steam disinfection.

In January of this year (1904), I visited three horsehair factories in Germany, to see the actual practice of steam-disinfection on manufacturing premises. In none of them was difficulty experienced in carrying this out; nor was it considered that the hair suffered in quality for manufacturing purposes, by treatment according to the Regulations. The occupiers were unaware of any hardship which had been inflicted on the trade by the requirement, nor had the price of the hair been increased by reason of the additional process. They expressed themselves satisfied as to the necessity of the measure, and believed its effect had been to diminish greatly the number of cases of the disease. One manufacturer, whose premises I did not visit as he was reluctant that I should do so, expressed an opposite opinion, believing that the industry had been hampered, especially for the small dealers (brushmaking at home), by the necessity of having to disinfect the material, or of having to purchase it from firms who had disinfected it. Further, he considered the hair was damaged by loss of its elasticity; but on further inquiry it appeared he employed a pressure of 28 lbs., instead of the 2¼ lbs. recommended in the Regulations.

In one factory a disused boiler had been converted into a disinfecting apparatus. In another, the walls of the disinfecting chamber were of thick wood, bound with iron bands. In the third, the apparatus was of iron—single-jacketed; the steam entered from above, and a valve prevented the pressure inside exceeding 2¾ lbs. Bales up to a weight of 100 kilos. were disinfected in bulk, if not hydraulically packed.

No disinfection is practicable in the case of hides or skins, and the Regulations which are now in force where hides and skins from China and East India are manipulated, only touch on such points as prohibition of

work for men with open cuts or scratches on exposed parts of the body, the wearing of overalls, and the posting-up of a notice detailing the symptoms: so that if the incidence cannot be lessened, the fatality of the disease may be lessened, as the result of prompt treatment.

That occasionally there is association of outbreaks of anthrax in animals with factory and workshop conditions must be admitted: as, for example, when the germs pass into water-courses and are there swallowed by cattle, or by refuse from woollen and horsehair factories being used on the land as manure.

It has, however, been asserted that incidence of outbreaks of anthrax in cattle is greatest in countries where industries connected with wool, horsehair or hides are mostly carried on. Such a statement as this seems, on the face of it, reasonable; but before it is accepted as a fact, the data on which it is based require close examination. To state merely the total number of outbreaks of anthrax among the animals in a county that have occurred is not sufficient. The number of animals—or at any rate of the cattle—in the county must also be known, because the incidence of anthrax must be worked out as a proportion on the number of cattle or animals in the several counties, in order to see whether the greater incidence in one county over another is not due merely to the greater number of animals in it. While most of the outbreaks have affected cattle, a fair number have also affected sheep, swine and horses; but most of the cases of anthrax in swine, I believe, have arisen, secondarily, from feeding the pigs on the carcases of cattle that have died of anthrax.

The Board of Agriculture publish yearly the number of reported cases of anthrax among cows, horses, sheep and swine in each county. The total number of these animals in each county is also stated. In view of the fact that anthrax in this country affects mainly cattle, it does not involve serious error, I think, if a comparative figure is obtained from consideration of—(1) the total number of outbreaks (1899 to 1903), and (2) the total number of cattle attacked (1899 to 1903), upon the total number of cattle in one year (1901). From reports kindly placed at my disposal by the Board of Agriculture, I have prepared the accompanying Table, which shows that no material difference is made whether the comparative figure is based on the number of outbreaks or on the number of cattle attacked. Comparison of this Table with the map showing distribution of the cases in the human subject

throughout the country, and also with the map showing the number of persons employed in wool, horsehair, and hides and skins in each county, negatives any sweeping conclusion that outbreaks of anthrax in animals are closely associated with manipulation of raw animal material in factories. Indeed, incidence on animals appears to be greatest in counties where danger from factory conditions appears most remote: as, for example, in Cornwall, Westmoreland, Aberdeen, Banff, Elgin, and Haddington. Generally speaking, incidence of anthrax in animals is twice as great in Scotland as it is in England, whereas incidence on the human subject is enormously greater in England than in Scotland. Incidence on the West Riding of Yorkshire is only fractionally greater (4.8 per 10,000) than the average for England as a whole (4.4). Incidence on Somerset is slightly greater than the average; but in Gloucestershire and Wiltshire less than the average. No doubt the direct influence of a particular factory in causing anthrax in animals must operate over an extremely limited area; and an unknown quantity in the production of anthrax among cattle is present in the extensive use as manure of refuse dust from wool and horsehair factories. Under the Woolsorting Rules, refuse dust drawn away by the fans in the sorting of dangerous classes of wool must be and is, burnt; and this is what probably happens to most of the dust removed under the Woolcombing Code; but it is known that dust from horsehair factories and felt factories is sold at a considerable profit, and that it forms an excellent manure for hop-fields.

The following case, where infection from the factory was direct, is interesting. Two valuable horses, grazing in a field on to which dust from Persian wool used in blanket manufacture was used, died from anthrax. Three or four cases among workers in this mill are included in the list of cases. On the sewage farm of a town in Worcestershire where incidence of anthrax on the workers from use of Persian wool has been excessive, there have been repeated outbreaks of anthrax among the stock raised on the farm. In 1903, a butcher in Derbyshire contracted anthrax after slaughtering a cow which had been pastured for a long time near a tannery. The animal was not known to be infected with anthrax; but another cow, which had been pastured with this one, developed the disease a few days later, and had to be destroyed. Here it was conjectured that infection was conveyed by the stream which runs through the works, and alongside the meadow in which the two cows were pastured.

Anthrax in Animals, 1899-1903, *in England.*

County.	Number of Cattle in 1901.	Total Number of Outbreaks, 1899-1903.	Comparative Figure of Number of Outbreaks per 10,000 Cattle.	Total Number of Cattle Attacked, 1899 1093.	Comparative Figure of Number of Cattle Attacked per 10,000.
Bedford	33,419	27	8.1	24	7.2
Berks.	43,608	9	2.1	12	2.8
Buckingham ...	73,390	14	1.9	13	1.8
Cambridge... ...	55,147	29	5.3	24	4.4
Chester	179,218	49	2.7	69	3.9
Cornwall	205,033	157	7.7	156	7.6
Cumberland ..	150,384	40	2.7	41	2.7
Derby	143,712	17	1.2	24	1.7
Devon	278,297	91	3.3	57	2.0
Dorset	86,675	41	4.7	52	6.0
Durham	77,797	8	1.0	6	0.8
Essex	86,097	39	4.5	39	4.5
Gloucester... ...	123,441	41	3.3	35	2.8
Hants	82,668	27	3.3	47	5.7
Hereford	92,522	17	1.8	19	2.1
Hertford	34,497	47	13.6	35	10.1
Huntingdon ...	28,794	6	2.1	9	3.1
Kent	73,047	62	8.5	38	5.2
Lancaster	240,484	181	7.5	192	8.0
Leicester	139,195	94	6.8	80	5.7
Lincoln	245,278	74	3.0	136	5.5
London	5,290	9	17.0	2	3.8
Middlesex	15,757	10	6.3	6	3.8
Monmouth... ...	48,270	7	1.5	10	2.1
Norfolk	143,543	53	3.7	8	5.6
Northumberland ...	121,190	142	11.7	125	10.3
Northampton ...	114,521	37	3.2	36	3.1
Notts.	83,447	32	3.8	29	3.5
Oxford	59,447	4	0.7	7	1.2
Rutland	18,205	11	6.0	12	6.6
Salop ...	179,160	32	1.8	42	2.3
Somerset	237,618	139	5.9	115	4.8
Stafford	165,888	46	2.8	84	5.1
Suffolk	78,276	24	3.1	21	2.7
Surrey	41,349	25	6.0	34	8.2
Sussex	112,069	45	4.0	51	4.6
Warwick	106,828	45	4.2	53	5.0
Westmoreland ..	70,226	82	11.7	89	12.7
Wilts.	113,275	46	4.1	39	3.4
Worcester	68,355	35	5.1	32	4.7
York—East Riding	86,683	40	4.6	54	6.2
,, North Riding	172,809	35	2.0	42	2.4
,, West Riding	276,626	132	4.8	136	4.9
Total ...	4,791,535	2,101	4.4	2,207	4.6

Anthrax in Animals, 1899-1903, *in Scotland.*

County.	Number of Cattle in 1901.	Total Number of Outbreaks, 1899-1903.	Comparative Figure of Number of Outbreaks per 10,000 Cattle.	Total Number of Cattle Attacked 1899-1903.	Comparative Figure of Number of Cattle Attacked per 10,000.
Aberdeen	180,927	397	21.9	471	26.0
Argyll	60,645	9	1.5	17	2.8
Ayr...	101,369	12	1.2	24	2.4
Banff	44,861	57	12.7	82	18.3
Berwick	16,905	5	3.0	5	3.0
Bute	9,512	1	1.1	1	1.1
Caithness ...	22,521	1	0.4	1	0.4
Clackmannan ...	3,685	--	—	—	—
Dumfries	63,974	14	2.2	24	3.8
Dumbarton ...	14,945	7	4.7	11	7.4
Elgin	22,671	49	21.6	64	28.2
Fife...	51,352	54	10.5	60	11.7
Forfar	55,772	91	16.3	122	21.9
Haddington ...	9,890	44	44.5	53	53.6
Inverness	51,483	2	0.4	5	1.0
Kincardine ...	26,497	21	7.9	24	9.1
Kinross	7,162	5	7.0	5	7.0
Kirkcudbright ...	50,361	4	0.8	5	1.0
Lanark	75,121	24	3.2	26	3 5
Linlithgow ...	12,366	6	4.9	8	6.5
Mid-Lothian ...	19,865	51	25.7	47	23.7
Nairn	6,247	3	4.8	14	22.4
Orkney	28,728	—	—	—	—
Peebles	7,304	1	1.4	1	1.4
Perth	75,900	66	8.7	101	13.3
Renfrew	26,997	7	2.6	16	5.9
Ross and Cromarty	44,367	24	6.3	30	6.8
Roxburgh	17,859	4	2.2	7	3.9
Selkirk	3,084	3	9 7	7	22.7
Shetland	19,050	1	0.5	3	1.6
Stirling	34,269	9	2.6	14	4.1
Sutherland ...	12,210	1	0.8	2	1.6
Wigton	51,382	35	6.8	42	8.2
Total ...	1,229,281	1.012	8.2	1,292	10 5

Successful actions have been brought by farmers against the occupiers of factories, for alleged loss of cattle from anthrax through pollution of streams with anthrax germs.

The influence of tanneries in spreading anthrax among the operatives and cattle on pastures watered by streams carrying off the refuse of tanneries was made the subject of inquiry by Dr. Ravenal, Bacteriologist to the State Live-Stock Sanitary Board of Pennsylvania. During 1897 he believes that 12 men and 60 head of cattle died of anthrax

near tanneries in that State. Each tannery had received part of a cargo of dry hides shipped from China, and no history of infection taking place after immersion of the hides into the tanning fluids could be obtained. This he attributed to the inability of a soft and flexible hide to produce a wound in the skin, as is easily caused by the ragged edge of a stiff, dry hide. He found no diminution in the virulence of the anthrax spores after immersion for long periods in the tanning fluid, nor could he suggest any efficient disinfection of the hides which would not injure them for further use.

Discussion on Dr. Legge's Paper.

The President: The Paper which Dr. Legge has read will provide, I am sure, ample field for discussion; and in order that we may get to it at once, I will reserve my observations until later, and will ask Dr. Arnold Evans to open the discussion.

Dr. Arnold Evans: I certainly think my first duty is to offer to Dr. Legge my personal thanks for the admirable manner in which he has approached this subject. We can hardly call this a Paper in the ordinary sense of the word: it is more in the style of an exhaustive treatise.

Most of the suggestions that I can make will be confirmatory of what has been already mentioned. On reading through the Paper, I noted one or two points upon which I should like to make a few remarks.

At the outset, I must say that I can only speak of wool. I have had no experience of anthrax occurring amongst those communities which are engaged in horsehair, bristles, or hides. But up in Yorkshire we have a large population concerned in the wool trade; and, if you look on the map, you will see that that big red splash represents a considerable population. Every year we have several cases: the numbers vary a good deal—sometimes we only have one or two in the year, sometimes 12, 15 or 20 cases of anthrax—and that affords a considerable field for investigation; and, as Dr. Legge has told us, it only happens amongst the workers in certain kinds of wool. The dangerous wools may be roughly classed as those which come from Asia. Asiatic wools are more dangerous than any other. Dr. Legge has told you that there is little or no danger from South American wools; but on that point I can

hardly agree with him, as I have known several cases amongst workers in wools imported from Buenos Ayres, and other ports of South America. However, no doubt the greater part of the dangerous wools came from Asia; we may take that to be so. The most dangerous as regards anthrax are, for some reason or other, wools coming from Northern India and the Central Asiatic States, Persian wool, and even mohair and wools from Northern India, certainly do contain the anthrax bacilli much more frequently than wools from any other part of the world. The greater part of the mohair (mohair, though called hair, is really wool) now imported into England comes from South Africa; but that is really a Turkish wool introduced into South Africa probably thirty years ago. The greater part of the mohair wool which is now imported into Bradford, although Asiatic in origin, yet is cultivated in Cape Colony.

There is another point in reference to the incidence of anthrax upon people engaged in the industry. In or about the year 1884-5, anthrax was recognised in Bradford as being of far too frequent occurrence, and the numbers of deaths were rapidly increasing. A meeting was arranged between the representatives of the workpeople and the representatives of the wool importers, and the medical officer of health; and in consequence of that meeting, a certain number of rules — or rather, suggestions — were drawn up, whereby the dangers from manupulating these wools were very considerably reduced.

Up to that time we had never seen or heard of the disease except among people who had followed the occupation of wool-washing or sorting, and for that reason it was known as "wool-sorters' disease;" but after the Home Office took this matter up, and issued their rules for wool-sorting, there seemed to be a shifting of the incidence of anthrax; and instead of attacking the wool-sorters in undue proportion, it largely affected those engaged in the further processes of combing and carding. Why that should be so I cannot tell; but I know that in some business firms a large amount of sorting has been done after the wool has been washed, and probably in some degree that might account for the incidence of anthrax falling upon those engaged in combing and carding than upon the sorters. However, such is the fact—that the disease is now more often heard of in the carding and combing rooms, and in consequence of that, the Home Office has issued special rules for combing rooms. Certain

it is, that even after wool has been washed and combed and carded it may still retain its infective properties. Only three weeks ago, I was informed by a "top-maker" in Bradford (perhaps I ought to explain that a "top" is really a wool which has been combed and made into great balls, which will then be ready for spinning into yarn. It is the long fine wool, as distinguished from the short wool. These tops are sent to the spinner and spun into yarn, which is sold afterwards to manufacturers for the purposes of weaving). This "top-maker" informed me that he sold some "tops" to a spinner in Glasgow, and he had been told that one of the workwomen spinners had been infected with anthrax; and he wanted to know whether I thought it possible that anthrax could have been conveyed to this person by means of wool which had undergone this process of washing and combing. I told him that I thought it unlikely; but afterwards the spinner himself came down from Glasgow, and said he had never imported any "tops" from Bradford before, and that he felt certain that the person had been infected by that. It seems very curious that after wool has undergone all those processes, it should contain the bacillus of anthrax in an active form; but no other cause could apparently be discovered for this girl getting anthrax, and certainly this wool was one of those which are classed by the Home Office as dangerous. It was Persian wool. So that I think there is something to be said in favour of the possibility that wool, even after undergoing all these processes, will yet retain the bacillus of anthrax.

Then, there is another point upon which Dr. Legge seemed to spend a considerable amount of time: as to whether or not anthrax might be communicated from the refuse of wool-combing establishments to animals. And here again we raise another point: Is the list of wools, scheduled by the Home Office as dangerous, complete? Are there not other wools which are not scheduled as dangerous, that are really dangerous in that they contain the anthrax bacillus also?

My experience rather goes to confirm the fact that wool, which is not scheduled as dangerous, may after all contain the bacillus of anthrax. I know a firm of woolcombers in Bradford who use the manure from the stables of their establishment, mixing it with the refuse which has been blown out in the sorting of the wool, and manuring fields with it at some distance from Bradford; and I found, some four or five years ago, when investigating an outbreak of

anthrax in cattle, in which three cases died, that the field in which these cattle grazed had been manured four or five months previously with wool from this woolcombing establishment. These people were supposed not to deal with dangerous wools at all, only with wools from Cape Colony and Australia. I think anthrax might be conveyed by the drinking of water. I had a case six weeks ago, in which two animals were found dead in a field, and the *post-mortem* and bacteriological examination demonstrated that they had died from anthrax. Well, they certainly had been drinking water from a stream which, probably half a mile or three-quarters of a mile above, had been polluted with washings from one of these woolcombing establishments. And there, again, they were not supposed to deal in dangerous wools: they worked only in wools from Cape Colony and Australasia. There is just a possibility that that water might have been infected by manure put on the fields. It does seem to me—in spite of the returns of the Board of Agriculture, which show that the prevalence of anthrax amongst cattle is not so great in these districts as in agricultural districts—it does seem that anthrax is conveyed by woolcombing refuse to animals. But I see nothing unusual in this: if a case of anthrax occurs in a herd of cattle in an agricultural district, I think it is much more likely to spread. You get there the effect of personal infection, and one knows that the sanitary conditions of cowsheds in rural districts are in some cases most shocking. Farmers seem to think their animals thrive better the darker, and the dirtier, and the more ill-ventilated their sheds are. They believe that in these conditions animals give more milk; perhaps they do, but they get tuberculosis —I know that.

In regard to the matter of disinfecting dangerous wools I have very little to say. On several occasions I have tried to disinfect dangerous wools by means of a Washington-Lyons Steam Disinfector: and although my disinfection may have been successful, the wool has almost always been irretrievably ruined, altered in texture and very much in appearance; and when I have shown it to the woollen manufacturer, he has invariably said that disinfection is no good at all; it may kill the bacilli, but at the same time it has irretrievably damaged the wool.

MR. ALBERT WEBB: There is one thing I should like to mention, which might perhaps explain the incidence of anthrax upon persons dealing with wool after it has been

washed. It is quite clear, of course, that even boiling will not destroy the anthrax spores, still less will simple washing do so: more particularly in the way that the washing of wool is often carried on at very low temperatures; in fact, such temperatures as would be just required for the propagation of anthrax. I have had a case where the person infected was dealing with nothing but material which had been entirely washed. Now I am pretty sure that, taking into consideration the comparatively low temperature, 80 deg. or 90 deg., at which washing is done, and the subsequent drying, that the spore is actually enlivened by the washing process rather than destroyed by it. That is the reason why anthrax appears after the wool has been washed. Washing clears off the dust from the outside; but it does not destroy the chance of the spores getting under the nails of the workpeople, and so getting into the body through that channel. The removal of the natural grease of wool is avoided as much as possible in the washing process.

Of course, wool is not my feature, and I do not know anything about it; but I know that in the case of horsehair, washing cannot be at all regarded as a safeguard against the spread of anthrax. We have had pretty certain cases of effluents from manufactories being drunk by cattle, and of anthrax being thereby conveyed to them. Washing, I think, does the spore good rather than otherwise, unless it is entirely eliminated. The spore is attached to the hair or wool by grease, by soil, and by animal discharges—blood, and so on—and washing does not readily remove these.

I have tested samples of dust extracted from hair by simply shaking the bundles, and the anthrax spore has been quite readily found present in them. I have then disinfected the bundles at a temperature of about 230 deg., and the dust then shaken from the bundles did not show anthrax. But if you took the same hair and broke it up, and so broke up the particles of grease, etc., which contain anthrax spores inside them, then you could get anthrax from the hair. So that, no doubt, the first thing is the keeping down of the dust; but you have by no means done with the matter when you have done that. In dealing with horsehair, at any rate, I maintain that it is not safe to handle these undoubtedly infected hairs—hairs from China, Siberia, and Russia—without some further process of disinfection, and that should necessarily begin as early as possible.

In my earlier experience we started with disinfecting in

bulk, in bales; subsequently in bags which were more or less open, and in bundles. But, eventually, although the first impression taken from the dust merely shaken from them was that disinfection had been complete, it became afterwards perfectly clear that the disinfection had not been successful. I subsequently found it necessary to thoroughly open the whole of the hair before sterilising with the Washington-Lyons apparatus. I used temperatures of 240 deg. to 250 deg. at first, but I found them by no means so successful as lower ones; later on, I used temperatures from 220 deg. to 230 deg., and these were undoubtedly better. If one gets down too low—simply to a condition of no pressure at all—the steam becomes too wet, and that will destroy the horsehair; but with a temperature such as the German authorities recommend, of from 2 lbs. to 3 lbs., I find no damage is done to raw material, and disinfection is distinctly better.

After having carried out for several years experiments of this kind—in this direction—I learned that another manufacturer, Mr. Carlo Pacchetti, of Milan, had also been making experiments as to the best method of dealing with the disinfection of horsehair. I wrote to him, and asked him if he would let me see what he was doing. He wrote to say that, although it was against the rule to allow other manufacturers to see trade processes, yet under the circumstances he would be very glad to give me all the information he could as to this matter. I went; and it was extremely interesting to me to find that, having experimented quite separately one from another, we had arrived at exactly the same conclusion as to temperatures, time and method, and were both using practically the same sort of machine. I had a Washington-Lyons apparatus, whilst he had a machine constructed in Germany, very much on the lines of the Washington-Lyons, except that it had an arrangement by which he could move the steam inside the chamber, which the Washington-Lyons has not. Mr. Pacchetti has had a great deal of personal experience; having in his employ something like 1,500 people. Apparently he has had a considerable number of cases; he had a laboratory there of his own, and kept his own analyst on the spot, with guinea-pigs and everything all ready for making experiments; he also had his own operating room.

In these experiments I have had the very valuable assistance of Mr. Cecil Duncan, County Analyst for Worcestershire; and I should here like to add a tribute of praise

to the County Council of Worcestershire, and especially to the Chairman, Mr. Willis Bund, who has done so much to facilitate the examination of this matter for many years.

It would be quite true, I am sure, to say that where any animals, such as horses or cows or sheep exist, that there anthrax will exist also, and therefore I can quite understand that anthrax has proceeded from other wools that those classed as specially dangerous. I am certain that anthrax exists in horsehair coming from South America; but there is a universal opinion throughout the horsehair trade in the world that South American hair is, speaking generally, not dangerous. Dr. Legge says here that there have been a certain number of suspicious cases, and I can quite believe that there must be cases which can be traced to South American hair. My agent in Buenos Ayres writes me that the disease is perfectly well known there under the name of "grano malo;" but the reason why it does not exist in so virulent a form is, that a great deal more care is taken with the animals. A man there who has a herd of horses, or a herd of cows, keeps his animals more or less under inspection; and if an animal dies under observation, that animal is buried with quicklime at once. Nothing of that description takes place in Russia or China, and there is no doubt that in those countries hair is collected from dead animals. The horse is a fairly long-lived animal naturally, but horses die in great numbers in countries where anthrax exists; and I have no doubt that many horses and other animals do die of anthrax; and that if the horse of a Mongol dies of anthrax, he will cut off its mane and tail, and sell it for importation to this country. It is the conditions by which the animal is surrounded that make the difference in the danger. The disease, as we know, exists everywhere: it exists even in England, in spite of all the care that is taken; but the conditions vary greatly in different countries. No doubt, the same considerations which apply to horsehair apply to sheep and goats, which produce the wool.

I have ventured to offer an explanation of the incidence of anthrax in wool, where it has already been washed and dealt with; and I think it may be necessary to examine this question further. How far wool may be treated by a disinfecting process, I cannot say; but I am quite sure that it is absolutely necessary altogether apart from washing: because, before we began with the Lyons Disinfector, we did boil hair in tanks, yet still we had cases of anthrax; so that I am quite sure that mere boiling is not sufficient to

thoroughly disinfect it. Since we have treated horsehair by disinfection, at a very low temperature—about 2½ to 3 lbs.—we have had no cases of it, nor have I found it in any of the hair itself. Although I have not experimented with wool, my impression is that wool would not be injured by steam disinfection at a temperature which was neither too low nor too high.

DR. HAMER: There are one or two points on which I should like to say a word.

First of all, with regard to the 30,000 cases in Italy which are mentioned, one wonders whether the term which is translated "anthrax" exactly corresponds with our word —whether the term used in Italy and our "anthrax" are mutually convertible. So large a number as 30,000 cases—of which 5,000 proved fatal—in ten years looks as if the Italian "anthrax" cases were made up of something more than the cases we know as anthrax: cases of carbuncle, phlegmon, or something of that sort; although one knows there is special risk in Italy of spread of anthrax, owing to the large number of tanneries which exist there. I should like to know whether Dr. Legge has any views on this point.

Then as to the statement on page 161:—

"The occurrence of cases in hides and skins in late processes, after immersion of the hides in the liming vats, points to the extraordinary vitality of anthrax spores; although, even in these cases, there is possibility of infection having been conveyed from untreated material."

My own impression is, that the second alternative is much more probable than the first. One has known a case where a man supposed to work only on tanned skins has contracted the diseases, and it has been shown afterwards that the man has been in contact with skins before their submission to the "liming" processes. There have also been cases amongst children going to school. Surely, it would be just as reasonable to attribute the infection to their having gone to school as it is to attribute the cases referred to in the tabular statement to "tanned hides," and so on. It must be that these persons, who were employed only in the later processes, had really been infected in some way which has escaped observation. Dr. Legge considered this probable in the case of the saddler which he has commented upon.

Again, as to the reference to the occurrence of internal anthrax in the hide and skin industry, made in the Report

which I prepared in 1894, I said I believed, as far as the evidence went, only one case had suffered from internal anthrax. Some of my critics were inclined to question there only being one case of internal anthrax. Therefore, it is some relief to my mind to find that after the lapse of ten years there are no further cases of internal anthrax forthcoming in the hide and skin industry.

Then there is the question raised in the final paragraph. Dr. Legge says that Dr. Ravenal "found no diminution in the virulence of the anthrax spores after immersion for long periods in the tanning fluid."

That seems to me to be a very important observation, if it is an accurate one. Mr. Spear suggested that the solution of the problem of prevention, in the case of hides and skins, was to be sought in some process by which spores could be converted into the more easily-destructible rods; and, of course, if there were some process of that sort, it would be of special importance, because there is such difficulty in disinfecting hides and skins. It seems to me, therefore, that the observation of Dr. Ravenal is worthy of some further notice.

MR. DEFRIES: I do not know that I have any but negative observations to make, beyond expressing the wish that industrial problems involving disinfection were more often attacked in as thorough a manner, and with such evidence of interest, in all parties concerned.

I have not anything very positive to say on this question, because I do not detect in the abstracts that Dr. Legge has given us any data which would enable us to know how the processes involved in disinfection were applied, *e.g.*, I do not know what methods alternative to the use of steam were used, nor what means were employed for controlling the penetration of the temperature—that is to say, of the steam—into the depths of the material under different conditions of press-packing. I see some question as to whether a uniform temperature was really attained at different points. It may be merely owing to the contraction that is inevitable in a Paper like this; but there are a variety of data that, on the facts as stated to us, undoubtedly want a good deal of investigating before we can be quite sure that we have got the last word as to steam disinfection. I am not quite sure that other kinds of disinfection, in which the action of steam was replaced by some chemical agency—I am not quite sure that that has ever been seriously tried at all.

Of course I am well aware—and it always strikes one first in a question of this kind—that it is unspeakably more satisfactory to prevent the possibility of access of infection than, having permitted the infection to come in, to turn to and see whether you cannot destroy it. At the same time it is not always possible to prevent infection. In this particular instance we are dealing with an imported article coming from countries, some of which have no mechanism even for enforcing regulations, and in none of which can your own authorities have any power, and imported infection must therefore always be liable to occur. It is practically impossible to prevent a Chinese gentleman from selling you anthrax bacilli as horsehair, if he can get you to buy them at the price of that article. Assuming that that is the condition of affairs, it seems to me that the case is one in which all supplies must be taken as liable to suspicion, and measures of disinfection applied as a matter of routine. Dr. Legge has called attention to the varying extent to which the anthrax infection imported from various countries seems to possess virulence. It does not seem to me that one is safe in inferring that the products of the countries which seem to furnish little infection are necessarily inoffensive. It occurs to me, for instance, that in anthrax there may be a secular variation of type, similar to those which I believe have been noted in other diseases. The variability of resistance of individual anthrax bacilli to disinfectant agencies is well known, and though not conclusive to this view, is consistent with it. There is, accordingly, no material predicting when a country furnishing little or no infection may lose its inoffensive character through secular change in the type of disease, or some other accidental variation in the conditions under which the product is obtained; and it remains a matter of the utmost importance to have means of disinfecting which will be applied as a matter of routine. If you have to injure the material, it is perfectly certain that you will not be able to get efficient disinfection; the administrative officers would have every man's hand against them, and the routine process would become a compromise in which disinfection ceases to be assured, occurring in a number of cases but failing in a certain proportion of cases. There is no evidence in the Paper and discussion that a trustworthy process is impossible. At present wool disinfection seems to be in a bad way. Yet Dr. Arnold Evans remarked that woollen manufacturers have said to him that disinfection of wool "almost always ruins the wool." What is the

meaning of "almost always"? I gather that sometimes the wool is not ruined. This varying result may, of course, be due to variations in the wool. It may, however, and perhaps more probably, be due to variation in the conditions of disinfection; and if so, then by defining experimentally and reproducing accurately the conditions which did not injure the wool, you would obtain a workable process.

The whole question of disinfection, not so much as regards horsehair—as to which the work has been at all events extremely thorough, and bears the appearance of complete accuracy as far as it goes—but in regard to wool, it seems to me to demand considerable further examination.

DR. BULSTRODE: I should like to ask Dr. Legge what is the relation of "Industrial Anthrax" to anthrax as a whole. We have heard to-night solely of "industrial anthrax," and I should like to ascertain, if possible, what margin there is left of the total notification figures for anthrax after "industrial anthrax" has been accounted for.

Dr. Legge has made a suggestion which is important to all epidemiologists, and that is that tanning processes do not destroy the spores or bacilli of anthrax: and looking at the resistant nature of the spores such destruction is problematical. There is, therefore, the possibility that the anthrax spores and bacilli may survive the biological processes of sewage purification, that they may get through these processes, and may give rise to epidemics of water-borne internal anthrax; but against this last supposition we have the testimony of experience that no such occurrence has ever been recorded, and we must take this fact into our consideration in summing up the position. We have, however, the fact that cattle have been infected from feeding upon sewage farms to which tannic refuse had access.

Also, if this bacillus or its spores survive, we should have expected to have heard something of shell-fish borne anthrax, but no suspicion of this has come beneath my notice.

There is another form of anthrax which I suppose would come within the margin which I am seeking to explain: that is, insect-borne anthrax. I have recently seen two or three instances recorded which are extremely interesting; and it is probable, I take it, that some cases of anthrax may really be insect-borne. How much I cannot say.

Then there is another question—the question of field-

borne anthrax among cattle. I should like to ask Dr. Legge how far his knowledge of the subject enables him to substantiate or refute the belief, which until recently was current, that the spores of anthrax might be conveyed to the surface by worms from the bodies of cattle dead of anthrax, and buried some feet below the surface. One is inclined to expect that the putrefactive organisms would destroy the pathogenic microbes long before they could arrive at the surface, or that the cattle would be buried so far below that there would be no worms to carry the spores on bacilli to the surface.

Then there is, finally, the question of what one may term Imperial-borne anthrax. I should like to ask how much anthrax is to be regarded as Empire-borne; that is, how much of it comes from the Colonies and from India, and how much from foreign countries. We are in a better position to insist upon proper precautionary measures within than without the limits of the Empire.

DR. WILLOUGHBY: Referring to Dr. Evans's remarks as to the spores not being killed, I should like to ask whether Dr. Evans has had any experience of the effect of formalin in disinfecting these wools.

Many authorities contend that the germs of putrefaction destroy the spores of anthrax in the putrefying carcase: a point of great importance in connection with tanning.

The German authorities do mention South America amongst suspicious countries; and they also speak of anthrax introduced in hay from Chili.

I should like to ask those two questions, as to the effect of formalin in disinfection of wool, and the question of putrefaction.

DR. PARSONS: There is one other trade in which cases of anthrax occur, *i.e.*, the trade of a butcher. Cases of anthrax are met with amongst butchers and people who have to do with the slaughtering of diseased cattle.

A case occurred in the neighbourhood of Manchester, not many weeks ago. A farmer noticed that one of his cattle was very ill indeed, and he apparently cut its throat to save its life, so to speak, and then sent to a butcher to dress the carcase, which was sold in the market. Both the farmer who slaughtered the heifer, and the butcher's man who dressed it, suffered from anthrax and died of it. The meat was all sold in Manchester, but it could not be traced. It was probably consumed for food, but no case of anthrax

was known to arise from it; although, of course, a case of anthrax might not be recognised if it occurred apart from industrial conditions. The only part that was not sold was the tongue, and that was put into pickle with seven other tongues, and those tongues were seized by the authorities, and submitted to Professor Delépine, who examined all of them, and in one of them found the anthrax bacillus. The butcher who had these tongues in his possession was had up and fined. It is somewhat remarkable that all this meat passed into the market and was consumed, and yet no harm seemed to have come of it. It is probable that in a carcase the anthrax is not in the spore-bearing form, and hence the bacillus may be destroyed by cooking. It is only when exposed to air that the spores form. The orders of the Board of Agriculture are very particular upon this point: they lay down that in the case of an animal that has died of anthrax, no cut is to be made in the carcase, so that the fluids containing the bacillus may not be exposed to the air, so as to form spores.

PROFESSOR KENWOOD: I have just one question I should like to ask of Dr. Legge. I understand from the excellent Paper he has given us this evening—which was most instructive and most valuable, I am sure—that in this country there are no regulations for the disinfection of horsehair, a product which is more particularly likely to give rise to anthrax; whereas in Germany there are regulations which, I take it, apply to all those who have to deal with this particular article. It seems that a current of steam, at a pressure of about 2¼ lbs., must be used in Germany, and that steam under this pressure is found to be sufficient to destroy the infection; and that, according to the experiments of the German authorities, it does not prove injurious to the material. That bears out the evidence which we have heard from the gentleman who spoke this evening, and who has had experience of horsehair, and its disinfection with steam, viz., that steam under a very low pressure is generally sufficient to destroy the infection of anthrax. I think that the prejudice against steam methods of disinfection amongst manufacturers in this country is largely due to the circumstance that it has been generally considered necessary to use a Washington-Lyons apparatus, or some other apparatus in which a pressure of from 10 to 15 lbs. of steam is used. Doubtless that may injuriously affect horsehair, and certainly wool; but there are experiments in this country, as well as in

Germany, which demonstrate the fact that the anthrax bacillus and its spores can be most readily killed at far less pressures (and lower temperatures) than that. Professor Delépine finds that in his current steam apparatus (which some of you may have seen), which was a laboratory apparatus in which the steam was current, and passed at a very low pressure through the apparatus, that every strain of anthrax bacillus and spores which he submitted to that steam were killed in two or three minutes. Of course, there is a great deal in what Dr. Legge says about the protection of the spores by particles of dirt and grease in the horsehair. In Germany, however, this current steam does appear to effect a considerable amount of good, judging from the statistics of the disease in Germany, and those of other countries.

I would ask, then, why it is not possible in this country to make and enforce regulations requiring the disinfection with low-pressure current steam of this very dangerous horsehair.

MR. DEFRIES: Professor Delépine's apparatus was a laboratory apparatus, which worked at a velocity of steam quite unattainable in an ordinary apparatus. I worked it out, and I found that it would have been quite out of the question to obtain anything like the velocity. If I remember rightly, Professor Delépine's Paper was directed to demonstrating that the efficiency of the apparatus varied directly with the velocity; but that velocity would be quite unrealisable in a commercially practicable machine.

PROFESSOR KENWOOD: I do not recall that Professor Delépine made that point, or that the experiments proved it exactly. He demonstrated the necessity for the steam to pass through the apparatus fairly rapidly, and he certainly put down certain conditions as to the outlet from the chamber having certain proportions to the cubical capacity of the chamber in order to effect this object.

THE PRESIDENT: Before asking Dr. Legge to reply, I want to tender to him on behalf of the Society our grateful thanks for the very admirable Paper he has given us.

DR. LEGGE: I shall answer Dr. Kenwood's question first. In the year 1898 only 3 cases, I think, were notified in the horsehair industry in this country; and it was then thought that the amount of anthrax in the horsehair industry was so much less than that in the woollen industry,

that regulations were unnecessary. The probability is that cases were occurring in the horsehair factories then just as they have occurred since; and that is the reason why I selected the five-year period we have been considering to-night, namely, because I believe that, dating from 1899, we do know of almost all the cases that have occurred. Then I am careful to say in the Paper that the German experiments were carried out with anthrax germs artificially introduced into the interior of the bales, and that they were in contradiction with the experiments of Mr. Webb and Mr. Duncan, of Worcester; and it was felt that regulations could not be applied to the horsehair industry of this country, until the whole matter had been completely thrashed out as to discrepancies in the results obtained.

So far as industrial anthrax, and cases which cannot be classed as industrial, are concerned in this country, I have no precise figures to give Dr. Bulstrode. During the five years in question, 38 cases have been notified to the Chief Inspector of Factories at the Home Office among butchers and farm-labourers and the like; but they cannot represent all the cases that have occurred. Personally, I do not think that anthrax in farmers and butchers has the same high rate of mortality which characterises anthrax in wool, horsehair, and hides and skins, and for this reason: that in most of them the pustule develops on the back of the hand or on the fore-arm, owing to direct inoculation from abrasions in the dressing of the carcase; and it is known that the mortality of cases where the malignant pustule occurs on the arm bears no relation to those which occur on the face. Professor Sclavo does not question the accuracy of the figures which I quote as having occurred in Italy. I have looked carefully through the 164 cases which were treated by Professor Sclavo—he gives a precise description of every one—and while some of the persons affected were employed in the industries I have dealt with (especially horsehair and tanning), still there are a considerable number of butchers, farmers, and the like, on the list. I think the figures quoted are reliable; I do not think they are confounded with erysipelas or other diseases.

I believe it has been proved that anthrax spores can pass through biological filter-beds; that the spores have been found in the catch-pit of a tannery, on the beds through which the sewage was passed, and again in the effluent. But Dr. Bulstrode is correct in saying that no case of internal anthrax from water or shellfish in the human subject is known. Anthrax the result of the bite

of an insect has been recorded in this country; and Dr. Williamson, of Cyprus, has shown that what is known there as "Sphalangi bite," sometimes develops into typical malignant pustule, the sting-bearing insect of the genus *Mutilla* conveying the virus.

Dr. Evans raised an exceedingly interesting point which has hardly been touched upon to-night, viz., why anthrax is largely confined to particular countries; and I cannot help thinking that it is possible there may be a difference of type of the disease in animals, as seems to be almost proved as regards anthrax in the human subject. Dr. Bell mentions 200 cases of malignant pustule in man treated by a surgeon in Argentina without a single fatal case: that does point in the direction of anthrax being much milder in South America than it is here. It is known that the disease is rife among cattle there, and yet not one of the cases discussed to-night can be attributed with any certainty to the very large quantity of material which comes from South America.

I think that if Mr. Defries had the opportunity of going through the papers published by the Imperial Health Office, he would find a detailed account of the methods taken by the German experimenters to assure themselves that there was penetration of steam into the interior of the bales. They experimented also with some chemical disinfectants, amongst others with formalin, and they were quite satisfied that they were inapplicable and ineffective for the purposes of the disinfection of horsehair. They found that these chemical disinfectants could not penetrate into the interior of the bale so as to destroy the spores.

With regard to the disinfection of hides and skins, I believe there is one preparation which, so far as laboratory experiments show, is effective in disinfection; but whether it could be carried out on a large scale commercially is a very different matter.

The Tables which form part of this Paper will show, in answer to Dr. Bulstrode's question, the proportion of cases of industrial anthrax which are attributable to English and Colonial material, as distinguished from that from foreign countries. Roughly speaking, all the cases in wool and horsehair in the last five years have arisen from manipulation of material from foreign countries, while one-half of the cases in the hide and skin industry were traced to material from India.

THE PREVALENCE OF ENTERIC FEVER IN PRUSSIA DURING THE DECADE 1892 TO 1901;

TOGETHER WITH REMARKS ON ITS ORIGIN, SPREAD, AND CONTROL.

BY DR. MORITZ PISTOR,

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(*Read: May 13th*, 1904.)

YOUR late distinguished President, Professor Corfield, my dear and honoured friend, invited me in January, 1903, to read a paper on typhoid fever to your illustrious Society, an invitation which I regarded as a high honour. I was the more willing to accept it as it offered me an opportunity of thanking English hygienists, medical officers and engineers, for all I have repeatedly learned from them in hygienic matters since 1869, when I first visited your sanitary institutions and public works. In agreement with Dr. Corfield, I chose for my address the following theme:—

"The Prevalence of Enteric Fever in Prussia during the Decade 1892 to 1901; together with Remarks on its Origin, Spread, and Control."

I was unable to read my paper earlier than to-day, owing partly to the fact that my official business demanded all my time and afforded me no opportunity for other work; and partly because I had decided, even then, to resign my office at latest on May 1st, 1904. But now, *medius in res.*

The name "Typhus" was known as far back as Hippocrates. In his time, and for centuries afterwards, that was the name given to any feverish condition attended with delirium, in which no organic disease manifested itself. But as knowledge increased, and the importance of the different symptoms was recognised, a distinction was made between the different conditions of disease, especially as post-mortems became more frequent, and made it possible for men to examine the appearances of disease,

together with the attendant organic changes. The first descriptions which appeared anywhere near the truth were written by a Dutchman, Willis, and by your fellow-countryman, Sydenham, who described a peculiar kind of *febris pestilens*, which raged in London between 1661 and 1664. Up to the beginning of the nineteenth century, no distinction was made between typhoid or enteric fever and spotted fever, which then went by the different names of petechial, famine, war, and ship typhus. Hildenbrand, in Germany, and your fellow-countryman, Murchison, were the first to draw attention to the differences between the two diseases, which could be recognised by the symptoms during life and the appearances after death. Rudolf Virchow, after his wide experiences, gained on the occasion of the great spotted fever epidemic in Upper Silesia in 1847-49, confirmed these theories; and he urged that the name "spotted typhus" should be abolished, in order to prevent erroneous ideas being current concerning the disease. The German imperial law of June 30th, 1900, relating to the combating of dangerous infectious diseases, has at length officially introduced the name of "spotted fever" for this disease, which is dangerous to the community on account of the rapidity with which contagion may be acquired.

I shall here speak only of the spread of enteric fever in the kingdom of Prussia, during the years 1892-1901, and of our experiences during this decade. These experiences throw critical light on the views held in Prussia at the present time concerning the origin, spread, and control of enteric fever. At the same time, I shall express my own views.

According to the four general reports for 1892-4, 1895-7, 1898-1900, and 1901, on the sanitary condition of the Prussian State, made to the Ministry of ecclesiastical, educational, and medical affairs, there occurred among every 10,000 living on January 1st of each year, the number of deaths shown in the accompanying tables. I give these surveys arranged in order, according to the average number of deaths in the administrative areas, with the exception of Sigmaringen,* which is too small to yield a fair average. But in doing this I must remark that the number of deaths officially reported does not correspond to the number actually occurring, and that it varies in each district, according as whether the sick persons have come under

* The district of Sigmaringen is an enclave of the kingdom of Wurtemberg, with only 66,700 inhabitants, and therefore not calculated to be of epidemiological value.

ADMINISTRATIVE AREAS

	1898.			istrative reas.		Enteric Fever Deaths per 10,000 Living. Ten Years, 1892 to 1901.
2.45	Treves	... 1.98	D	...	...	2.62
1.96	Minden	... 1.78	A	...	...	2.32
1.82	Erfurt	... 1.77	T	...	...	2.29
1.81	Marienwerder	... 1.74	K	rder	...	2.11
1.80	Arnsberg	... 1.73	S		...	2.03
1.79	Gumbinnen	... 1.62	M		...	2.01
1.78	Stettin	... 1.60	E	..	...	2.01
1.76	Danzig	... 1.54	B	g	...	1.94
1.74	Magdeburg	... 1.52	H	n	...	1.87
1.74	Königsberg	... 1.50	K	..	...	1.78
1.64	Köslin	... 1.38	S	..	...	1.74
1.61	Bromberg	... 1.30	G	g	...	1.57
1.54	Koblenz	... 1.23	O	..	...	1.56
1.49	Liegnitz	.... 1.19	D	n	...	1.54
1.46	Merseburg	... 1.15		..	...	1.52
1.45	Staat	... 1.14	M	sen	...	1.46
1.39	Münster	... 1.12	C	..	...	1.45
1.32	Osnabrück	... 1.07	K	..	...	1.44
1.30	Hannover	... 1.07	L	..	...	1.39
1.28	Breslau	... 1.06	P		...	1.35
1.24	Posen ...	... 1.04	M		...	1.33
1.17	Düsseldorf	... 1.03	A	..	...	1.30
1.16	Frankfurt	... 1.01	H	..	...	1.29
1.13	Hildesheim	... 1.01	B		...	1.28
1.10	Aachen	... 0.92	F		...	1.26
1.09	Cöln ...	... 0.90	M	..	...	1.22
0.99	Stade ...	... 0.89	L	..	...	1.20
0.96	Potsdam	... 0.86	P		...	1.15
0.91	Wiesbaden	... 0.84	S	..	...	1.15
0.90	Stralsund	... 0.78	C		...	1.13
0.87	Cassel	... 0.76	M	..	...	1.12
0.82	Sigmaringen	... 0.76	S		...	1.09
0.82	Schleswig	... 0.74	O	..	...	1.06
0.76	Oppeln	... 0.72	W		...	0.85
0.76	Lüneburg	... 0.66	B	..	...	0.72
0.65	Berlin	... 0.56	S	..	...	0.63
0.53	Aurich	... 0.47	A			

medical treatment, and whether the physicians have fulfilled the duty of notification prescribed by law. For these reasons, we must give up any idea of calculating the number of cases.

The evidence at hand enabling us to form any judgment of the behaviour of enteric fever in Prussia is by no means to be relied on. Nevertheless, a ten years' survey of the number of deaths reported enables us to form an approximate idea as to the prevalence of the disease in the different districts and in the whole State, and justifies us in the assumption that enteric fever is, on the whole, decreasing. According to the general sanitary reports of 1895-97 (p. 76), the mortality from enteric fever since 1875 has been inquired into in the same fashion; and calculations have been made, with the result that it is found that in the whole State the mortality has receded from more than six to less than two deaths per 10,000 living.

In order to present to your mind a clearer picture of the prevalence of enteric fever in the different parts of the State, I have endeavoured by means of different shadings, to represent on the map, to be found at the end of this Paper, the number of deaths in the different districts per 10,000 living, according to a ten-year average.

I shall not enter into the question of the proportion of different ages and sexes in the mortality, chiefly for the reason that these numbers usually remain about the same in the course of the year, and have little value as regards the epidemiological and hygienic discussion of the enteric question.

If you examine the statistical tables of deaths in connection with the various shadings on the map, you will notice that neither the greater prevalence of the disease, nor the decrease in its occurrence, depend on conditions of climate, or on geographical or geological limits. In 1892, 1893, 1895, 1896, 1897 and 1898, the administrative district of Treves, lying on the Mosel, close to the French frontier, shows the highest mortality; whereas simultaneously or uniformly the districts of Stralsund, on the Baltic, Danzig, on both sides of the Vistula, Bromberg, lying on the borders of Germany and Russia, Marienwerder, Gumbinnen, also the inland district of Arnsberg, and the district of Erfurt, situated in the heart of Germany, near the Thuringian Forest—show nearly the same degree of mortality: that is to say, more than two deaths from enteric fever per 10,000 living. In fact, practically the same districts show the same high death-

rate, even taking a ten-year average. Next to the western mountainous district of Treves, situated on the volcanic soil of the Eifel Mountains, and having the highest death-rate: namely, 2.62 per 10,000 living, comes the flat Danzig district, in a very fertile plain, and having an oceanic climate. Treves has hot summers and mild winters; Danzig, a more equable climate, with colder winters. The district of Arnsberg—with its widespread industry, above and below ground, lying in mountainous country, richly wooded in parts, in the province of Westphalia, between the Weser and the Rhine, and having a continental climate—takes the third highest place among the portions of the State most invaded by the disease. In 1901, owing to the widespread Gelsenkirchen epidemic, it takes the highest place of all the districts. Next comes Marienwerder, a district almost entirely devoted to agriculture, and situated on the eastern side, close to the sea, and, like Bromberg, bordering on the Danzig district. Equal to it stands the district of Erfurt, as in former years. There still remain eight districts above the States ten-year average death-rate of 1.46 per 10,000. These are the flat districts of Königsberg, Gumbinnen, Stettin and Köslin, lying east of the Elbe, and chiefly devoted to agriculture, and the districts of Magdeburg, Aachen, Coblenz and Hildesheim, lying west of the Elbe. This explains how, according to ten-year averages of deaths officially reported, enteric fever has prevailed on the one hand in the western mountain districts of Treves and Arnsberg, having a continental climate and widespread industry, especially the Saarbrück coal-bed and Westphalia coal-district; and on the other hand, in the district of Stralsund, engaged chiefly in agriculture, lying near the Baltic, and having an oceanic climate; also in the West-Prussian province (Danzig and Marienwerder), and in the East-Prussian province (Königsberg and Gumbinnen) similarly situated, and having an almost similar climate. The districts of Coblenz and Hildesheim appear as moderately-infected enteric islands, in the midst of large portions of the country but little attacked. Perhaps Coblenz suffers on account of its proximity to the severely-affected district of Treves, but Hildesheim borders on no severely-affected Prussian district.

If we now consider those districts in which the ten-year average death-rate falls below the average of the State, the most striking feature is, that the provinces of Schleswig-Holstein (bounded by the North Sea and the Baltic, having an oceanic climate, and inhabited by people engaged almost

exclusively in agriculture) and Hesse-Nassau, with a continental climate, lying in the heart of Germany, bordering on the severely-affected administrative district of Arnsberg, and having likewise a population engaged for the most part in agriculture—remain far under the ten-year average mortality of the State. We notice also that in the administrative area of Wiesbaden less than one death occurs per 10,000 living. In the province of Silesia, which has a continental climate, Lower Silesia, inhabited chiefly by peasants as far south as on a line with Breslau, exposed on all sides except the north to the introduction of all the indigenous infectious diseases of Europe, from Russia and Austria-Hungary, stands under the average enteric fever death-rate for the State. Indeed, the administrative area of Oppeln—which, as far as hygiene is concerned, is in ill-repute specially for the spread of infectious diseases, and which has a widespread industry above and below ground—has the lowest ten-year average death-rate in the province, far under the average of the State. With its ten-year average of 1.12 per 10,000, it does not equal the half of the Treves and Arnsberg averages, although these two districts have a similar inland position, similar conditions of climate, and industrial conditions almost identical with those of Oppeln, the southern part of which has constant intercourse with Russia and Austria-Hungary, through workmen constantly passing to and fro.

The districts of the province of Hanover, with the exception of the district of Hildesheim, remain very far below the State average; in fact, the administrative area of Aurich, which borders on and partly lies in the North Sea itself (*e.g.*, the Frisian islands, Norderney, Borkum, etc.), has the smallest average mortality in the whole State, namely, 0.63.

The Rhine province and Westphalia, with the exception of the administrative areas of Treves, Coblenz and Arnsberg, stand far below the average of the State. Likewise the province of Brandenburg, not including Berlin and the administrative area of Merseburg. These portions of Prussia have conditions of climate fairly similar to those of Magdeburg and the districts of the province of Hanover. They are, too, chiefly agricultural districts, lying on the same soil of the North German lowland as the provinces of East- and West-Prussia, Posen and Pomerania. After the administrative area of Aurich, the imperial capital and the administrative area of Wiesbaden have the lowest enteric fever mortality.

The spread of enteric fever in the districts of Treves, Arnsberg and Oppeln is favoured by several circumstances common to all three. The hygienic conditions are in many respects unfavourable. In the industrial portions, the mining industry has drained off the water by its mines. The wells they have are either dried up or give bad water, dangerous to the health, whether used for drinking or ordinary domestic purposes. In the coal district of Westphalia, and in Upper Silesia this evil has hitherto been compensated for, on a large scale, by central water supplies. The water services in the administrative area of Arnsberg supplied bad water by making use of unfiltered water, from time to time, from the river Ruhr, as was incidentally shown in the case of the epidemic of enteric fever in the urban and rural district of Gelsenkirchen, 1901-1902, according to the report of the Government Medical Officer, Dr. Springfeld, in the tenth volume of the "Clinical Year Book," (published in Jena by Gustav Fischer, 1903, p. 323). On the other hand, the industrial district of Upper Silesia has in parts, since 1897, had a plentiful supply of drinking-water—good in every respect—whereas the district of Treves has only a small number of central water-supplies above suspicion. For drinking and domestic purposes, the inhabitants for the most part use surface water from very inadequate open draw-wells, or covered pump-wells, which are contrary to the regulations. Mention must also be made of the indifference on the part of all classes of the working population (especially of the miners) to the danger of infection, which indifference often turns into passive resistance against the official regulations. And together with this comes the crowding of the population, and the small inclination which the people possess towards good order and cleanliness. After former experiences, there can be no doubt that want of cleanliness promotes the spread of enteric fever. Moreover, the practice of constantly depositing human dejections and household sewage in the water-courses, where the soiled linen, as well as the chamber-utensils used by the enteric fever patient are also washed, favours the spread of the disease: as it is a matter of experience that the inhabitants often take water for drinking and domestic purposes from these same water-courses.

The question is now, how to explain the fact that enteric morbidity and mortality is so great in Treves and Arnsberg and so small in Oppeln.

In these three administrative areas enteric fever is most

widely spread in those districts which are almost entirely engaged in industry above and below ground. The inhabitants are chiefly workmen, who in the Arnsberg industrial district form 60 per cent. of the population: for the most part foreigners, Poles, Italians, and even Roumanians and Walloons. In both Upper Silesia and Treves the inhabitants have similar manners and customs. Owing to their birth and education, they have no idea of order and cleanliness. The dwelling-houses are for the most part mean and small, certainly in many cases too overcrowded: a natural result of the rapid increase of the working population in those districts, together with the extremely flourishing condition of the industry since 1870. The Table which is reproduced overleaf will serve to show you the increase of population in the whole Prussian State during the decade 1890 to 1900.

You will now be able to form some conception of the rapidity and great extent of the increase of population in the industrial districts, and of the exceedingly small increase in the agricultural districts.

For the sake of illustration, I shall only draw attention to the difference between the increase of population in the administrative areas of Oppeln and Liegnitz. The district of Breslau, which lies between the two, cannot be compared, because the town of Breslau, with nearly half a million inhabitants, outbalances the comparison. Further, the results of the great increase in the industry are to be seen in the industrial portions of the districts of Arnsberg and Treves, which have a closely-crowded population. Nevertheless, these conditions cannot be made exclusively responsible for the spread of enteric fever, since, for example, the Marienwerder and Bromberg districts, situated close to the boundaries of Russia and Poland, and but thinly populated, as also the districts of Stralsund in cleanly Pomerania on the Baltic, and Erfurt in the heart of Germany, stand far above the average mortality of the State, which is 1.46 per 10,000 inhabitants (see Table facing page 200).

From personal observation during seven years' official work (1874 to 1881), I am well acquainted with the local and other conditions of the Oppeln district, which are said to have altered but little during the last thirty years. Consequently, I cannot help thinking that, as enteric fever in the industrial district has up to the present never altogether died out, the small number of deaths from that disease during the last ten years must be attributed to

the apathy of the inhabitants, who seldom call in medical aid, and in the event of a death occurring, usually give the registrar the name of some favourite disease as the cause of it. Whether the doctors there perform their duty

Density of Population in Prussia, 1890, 1895, *and* 1900.

Districts.	Average Number of Inhabitants per Square Kilometer.		
	1890.	1895.	1900.
Königsberg	55.53	57.05	57.06
Gumbinnen	49.54	50.52	49.87
Danzig	74.09	77.71	83.70
Marienwerder	48.08	49.88	51.07
Berlin	24,906.04	26,460.07	29,816.07
Potsdam	68.05	80.05	93.48
Frankfurt	59.24	60.93	61.43
Stettin	62.03	65.02	68.77
Köslin	40.18	40.95	41.89
Stralsund	51.94	53.46	53.94
Posen	64.33	66.99	68.40
Bromberg	54.59	57.23	60.17
Breslau	118.64	121.48	125.91
Liegnitz	76.97	78.42	81.04
Oppeln	119.35	129.34	141.25
Magdeburg	93.13	97.52	102.18
Merseburg	105.36	110.61	116.53
Erfurt	122.67	126.52	132.07
Schleswig	64.41	67.70	73.03
Hanover	92.06	102.23	113.33
Hildesheim	89.59	93.01	98.43
Lüneburg	37.04	39.31	41.66
Stade	49.83	52.09	55.26
Osnabrück	48.27	50.34	52.96
Aurich	70.19	73.39	77.24
Münster	73.94	81.97	96.45
Minden	104.55	111.43	121.07
Arnsberg	174.46	197.60	240.54
Cassel	81.47	84.40	88.29
Wiesbaden	150.22	161.35	179.42
Coblenz	102.12	104.85	109.97
Düsseldorf	360.55	400.41	475.02
Cologne	207.96	227.68	259.42
Treves	99.12	106.97	117.03
Aachen	135.89	142.03	148.00
Sigmaringen	57.86	57.56	58.46
State	85.97	91.38	98.87

of notification better or worse than in other districts, or whether, since my leaving Upper Silesia in 1881, the general conditions of health have so improved as to be able to account for the decrease in enteric fever, I am not in a

position to judge. We must leave it to the future to show whether new widespread epidemics will find a resting-place there, or remain absent.

I will now, with your permission, throw light on the spread of enteric fever in the agricultural districts most severely attacked, namely, the administrative areas of Dantzig and Marienwerder in the province of West-Prussia. the administrative areas of Königsberg and Gumbinnen in the province of East-Prussia, and the administrative area of Stralsund in the province of Pomerania. These must be compared with the province of Schleswig-Holstein, the administrative area of Wiesbaden, and the greater portion of the province of Hanover; in particular the administrative area of Aurich.

The imperial capital, Berlin, cannot be brought forward for comparison, because the conditions of a large town differ too widely from the conditions of small or average-sized towns, and all too much from those of flat country. Through the prosperity of its inhabitants, and by reason of the revenue from its extensive possessions, Berlin, with the advice of the best experts, is in a position to take all measures which can help to improve the public health. By the introduction of a complete system of sewerage in 1875; by the continual improvement of the drinking-water for all parts of the town; by various measures which promote cleanliness; by the erection of dwellings which more and more tend to the preservation of health; by an ever-increasing watchfulness over the sale of articles of food; by all these measures the local administration of Berlin has undoubtedly improved the conditions for the health of the inhabitants in a most praiseworthy manner. The best proof of this is the ever-decreasing number of enteric fever cases and deaths. This disease manifestly thrives and spreads rapidly on polluted soil, among a densely-crowded population. Berlin, therefore, also from the standpoint of public health, holds an exceptional position compared with the other districts of the kingdom of Prussia.

As causes of the origin and spread of enteric fever in the provinces of East- and West-Prussia, I must mention the introduction of the disease by patients brought in from invaded districts; the pollution of the water-courses by all kinds of sewage, liquid sewage from dung-middens and cattle stables, and human dejections, which make their way into the water either directly or indirectly through the washing of linen, into the wells, water-courses, or even central-water mains: lastly, infected articles of food,

especially milk. From 1897 onwards, the theory of the transmission of the disease from person to person by direct or indirect contact (contagion), here as well as in the reports from other districts, has again found more supporters. For views, like fashions, change according to the conception of the different leaders, who are often followed and quoted without a thorough investigation of and deeper reflection on the statements they make.

If we but consider the circumstances somewhat closer, we see, firstly, that in both provinces the working classes of the population who, it is true, live much more scattered in the country than in the industrial districts, on which judgment has before been pronounced, live, however, just as closely crowded together in the dwelling-houses as the workmen of the industrial districts; secondly, that dirt is held in just as much honour by this population, in parts composed largely of Poles, as by the people of those other industrial districts; thirdly, that the people here are just as careless in the disposal of excrement as they are there; fourthly, that disregard of danger is just as great here as there. In short, conditions which in my estimation everywhere favour the origin and spread of enteric fever, after enteric germs have once been imported, exist here, as they do there. The administrative area of Stralsund alone forms an exception. For it is just in New Hither Pomerania that the inhabitants are remarkable for their sense of cleanliness, which is considerably greater here than it is among the inhabitants of Posen and East- and West-Prussia. The water-supply of the two larger towns, Stralsund and Greifswald, has for years been above suspicion. There is there no mention of epidemics spread by milk infection; but the faulty condition of the wells in the country districts is, without further proof, brought forward as a probable cause of the cases of enteric fever. In my opinion, the reason of a higher mortality being officially reported in the Stralsund administrative area may be found in this, namely, that here a mortality relatively greater than in other districts is reported to the authorities, because cases from country places are taken to the University Hospital in Greifswald, and notified from there. The same thing happens from the local administrations of the numerous town and district hospitals of this small district.

The high average mortality for the districts of Bromberg and Erfurt, as compared with the administrative area of Posen, where in all points favouring enteric fever con-

ditions are the same as in the district of Marienwerder, can in my estimation only be explained by the fact that in the Posen district similar faulty notification of cases and deaths occurs here as in the case of the Oppeln district.

If we now turn to the districts with the lowest ten-year average mortality (excluding the imperial capital and the district of Sigmaringen), we see that we must include the province of Schleswig-Holstein, almost the whole of the province of Hanover, Hesse-Nassau, and a large part of the Rhine province. The inhabitants of the province of Schleswig-Holstein distinguish themselves from the inhabitants of other portions of the State districts by the cleanliness which exists even among the working-classes, and which is specially noticeable in their dwellings and surroundings. There is also a general prosperity among them, and the province is not too thickly populated. The same holds good of the East Frisian portions of the province of Hanover, in the administrative areas of Aurich, Stade, Osnabrück, Lüneburg (with the exception of the manufacturing town of Harburg) and the district of Hanover; whereas in the Hildesheim district the prosperity and cleanliness of the mountain population is not so great.

In the districts of the Rhine province and the province of Westphalia, which are not so much infected by enteric fever, it is possible that greater appreciation of cleanliness and greater prosperity may have limited the spread of the disease. But we must also take into account here, as in the case of the provinces of Hesse-Nassau and Brandenburg, that there is a much smaller influx of foreign workmen, who, more often than is generally assumed, introduce enteric germs through the medium of sick persons, and persons in apparently good health. The following fact, among other things, is not to be overlooked, namely: that there is very little apathy on the part of the general population, which can be traced back to the most ancient German culture of those districts. I am not aware that those districts attacked only slightly are in general supposed to have made greater efforts in the direction of promoting public health, than those districts attacked more severely.

Now, what attempts have in the course of time been made to explain the origin and spread of enteric fever?

In this gathering of scientific and practical experts, I need do no more than merely remind you of the principal views held as to the origin and spread of enteric fever up to the end of the nineteenth century; and I can omit the

mystical ideas of the Middle Ages, when men still looked upon this and other diseases as judgments from heaven for sins committed, and tried to banish them by prayers, etc.

During the 'sixties of last century, Murchison, in his "Treatise on the Continued Fevers of Great Britain," upheld the autochthoneus origin of enteric fever, and based his theory on the fact that the disease occurs more especially in those houses situated on a subsoil polluted with the putrefaction and decomposition of organic matter. This theory met with much approval; but, on the other hand, it was pointed out that the appearance of enteric fever was, as a general rule, connected with certain seasons of the year; and at the same time this seasonal theory was placed in opposition to, or alongside of, the localisation or soil theory.

Through Buhl and von Pettenkofer, of Munich, the so-called ground-water theory sprang into existence, according to which enteric fever stands in a certain relation to the fluctuations of the level of the ground-water. Buhl summed up his theory as follows: He considered that, undeniably, a relation existed between the fluctuations of the level of the ground-water and the extent and intensity of enteric fever, in the following way, namely: that the total number of cases and deaths decreased with the rise of the ground-water, and increased with its fall. However, the height reached by the disease was not in proportion to the level of the ground-water in each case, but only in proportion to its movement in each case. In other words, it was not the higher or lower level of the ground-water which was of decisive importance, but merely the amount of fluctuation. In this way also it could be explained how there came to be an increase in the spread of the disease during the summer months, when the ground was dried up, and a decrease during the winter months.

It was upon this that von Pettenkofer and his followers founded their well-known localisation theory, according to which the origin and spread of enteric fever was said to be dependant on local, seasonal, and personal conditions. But not only was it stated that the infective matter resided in the soil, but it was also said to there undergo a maturing process, which rendered it particularly virulent, in a manner analagous to cholera. These views at first met with great approval. It was thought that the origin of the infective matter—the miasma—was now known, and it was assumed that the miasma was spread by the air, and that the disease

was also transmitted by direct contact of a patient with a person in good health.

But as far back as the 'sixties of last century, all investigators of that time, at the head of whom stands your fellow-countryman, Budd, expressed the view that the infective matter was probably a *contagium vivum*. This view was confirmed in 1880, by Eberth's and R. Koch's discovery of the enteric fever bacillus. There is no time to go into the details of this discovery and its further hygienic development, particularly by the careful work of Gaffky and others. Besides, gentlemen, the details of the investigation, together with its results, will be better known to you than to me: a public official advanced in years, whose duty is not the scientific investigation of epidemic diseases, but the participation in the fight against them, based on the results of scientific investigations. It is in this light that I beg you to regard the following observations.

Enteric bacilli are aerobic rod-like microbes, 1—3 μ long, and 0.5 to 0.8 μ broad, with rounded or flattened ends. They easily stain with aniline dyes, but never by Gram's method; they show a fairly strong mobility in the hanging drop, associated with the peculiar flagella of the bacilli. They grow easily on different media, especially on potatoes and agar, in water, bouillon, milk and whey, and on other articles of food, blood-serum, and such-like materials.

Further investigations of the behaviour of enteric bacilli were made, with the object of discovering a sure means of distinguishing them from bacilli of an enteric-like nature, particularly the bacilli of the *Coli* group. R. Koch, by his discovery of the bacteriological media, has the honour of having shown the way to distinguish between the different bacteria. Jointly with Issaeff, Kolle, and A. Wassermann, Pfeiffer was the first to utilise the specific characteristic of the enteric-fever immunising serum, for the purpose of distinguishing the enteric bacillus in the disposition of the so-called Pfeiffer reaction from bacteria of an enteric-like nature. Max Gruber and Durham, equally important men, build on these fundamental experiments, discovered the agglutination test, and recommended its use for the differential diagnosis, or identification of the enteric bacillus. Further, Widal proposed the utilisation of the agglutination test for the early diagnosis of enteric fever, and the value of the Widal method was soon proved by Grünbaum*

* After reading this Paper at the meeting of May 13th, I found in the *Lancet* of February 1st, 1902, a correspondence, by which it is evident that Dr. A. S. Grünbaum in Liverpool, already in March, 1896, used the

in Liverpool, Mesnil, and C. Fraenkel, in the Koch Institute for infectious diseases, and in many other places.

This agglutination method of Widal, I may briefly add, consists in taking diluted blood-serum in a certain proportion (1:40 to 50, to 100) from a patient with fever, suspected to be enteric, and adding a young enteric culture. If clumping (agglutination) takes place in the mixture, it means that in all probability the case is one of enteric fever. Nevertheless, agglutination is also occasioned by some other kinds of bacilli, even by bacilli from cases not of an enteric-like nature; and lastly by bacilli of paratyphoid fever. When the Widal reaction turns out negative, it does not show that there is no enteric fever present. Lastly, it must not go unmentioned that, for a month after recovery from enteric fever, when the enteric bacilli still remain in the human body, repeated attacks of fever may occur. These may also be looked upon as enteric fever.

Quite lately, in the *Berliner Klinische Wochenschrift* for 1903, No. 45, M. Ficker has mentioned a simplified Widal method, in which a substituted liquid is used, which gives a reaction without the use of living enteric cultures. According to the information *loco citato*, it should meet all requirements, but we are still awaiting Ficker's detailed report. So far as I am aware, we do not yet possess an infallible method for the bacteriological investigation of enteric fever, like the Peptone method for cholera, We await the confirmation of the results obtained. Therefore, the value of the Widal reaction, as of all bacteriological investigation, must always be estimated in association with the clinical symptoms, if we wish to some extent to avoid false diagnosis; under all circumstances, a suspected case of enteric fever must be regarded as such, and accordingly prophylactic measures applied until the contrary is proved.

In difficult cases the bacteriological investigations must extend to the secretions and excretions of the body, as well as to the blood. The methods are often very complicated, and cannot be entered into here. Reference must be made to the manuals and hand-books on bacteriology. Meanwhile, I cannot leave unmentioned Conradi's and von Drygalski's test for enteric-fever bacilli, seeing that, according to R. Koch, it held good in the discovery of latent cases

agglutination for the diagnosis of enteric fever in the laboratory of Professor M. Grueber in Vienna. M. Grueber announced this fact at the meeting of the Medical Congress at Wiesbaden in April, 1896.—M. P.

of enteric fever in the administrative area of Trier in 1902. A full and detailed description is to be found in vol. xxix of the *Zeitschrift für Hygiene und Infektions-krankheiten*, p. 283 ff. Here I can only furnish an extract.*

Substances are added to litmus-agar medium, which, instead of preventing the growth of enteric-fever bacilli, favour it as much as possible, and inhibit the growth of all intestinal bacteria, with the exception of *coli*-bacilli, which remain behind with the enteric bacilli, together with some other unimportant microbes. *Coli*-bacteria form much acid; enteric bacilli, on the other hand, but little. In the event of correct lowering of the reaction of the culture medium, it is now possible by the addition of a litmus solution to bring it about that the enteric colonies remain blue, whereas the *coli*-bacteria turn red. For the further testing of the enteric bacilli, the agglutination test is used, and investigation made in the hanging drop. But the serum used must be thoroughly trustworthy.

By this method a reliable diagnosis can, according to R. Koch, be obtained in twenty to twenty-four hours, and in a much earlier stage of the enteric fever than is possible by the use of Widal's method. But at the same time we must always keep in mind that the Conradi and von Drygalski method

1. Is no improved method;
2. That, according to previous experience, it not infrequently leaves us in doubt in the early stages and at the height of the disease;
3. That the carrying-out of this method requires special practice.

Thus enteric cases owe their origin solely to the enteric bacillus; but the spread of the disease is specially favoured by the bacillus falling on a fruitful ground. Spontaneous origin is, according to the previous investigations, out of the question; neither is it any longer possible to speak of the miasma, which in former times people believed in. Therefore, statements from different parts of the Prussian State to the effect that enteric fever owes its origin to soakage from dung-heaps, to contamination of the air, water-courses, wells and soil, are to be rejected; in so far that it cannot be proved that enteric bacilli gained an entrance to those places, or at least had any probability of gaining an entrance. On the other hand, it follows un-

* R. Koch's Paper, "Die Bekämpfung des Typhus. Vortrag, gehalten in der Sitzung der wissenschaftlichen Senats bei der Kaiser Wilhelm Academie am 28 November, 1902. Vorlag von A. Hirschwald. Berlin, 1903." See p. 11.

doubtedly, from the general sanitary reports which are taken as a basis, that when the enteric fever bacillus is once disseminated, it finds that ground most suitable where cleanliness is wanting, where pure drinking-water is lacking, and where the people are closely crowded together, particularly in the houses, and do not understand the dangers which arise from neglecting the laws of public health.

Without further proof, it is evident that the greatest and most rapid spread of enteric fever must occur when a larger number of persons are simultaneonsly exposed to the same carrier of the germ of infection. This is brought about most easily and is most widely spread, when the source of the general water-supply of some district, or of a number of dwelling-houses, is polluted by enteric fever bacilli. This knowledge is no achievement of more modern times: it was known as far back as the middle of last century. I remember that the well-known Dr. Bamberger, Professor of Clinical Medicine in Würzburg, traced an enteric fever epidemic of 1856, confined to the vicinity of the University there, to the drinking-water of a well, and at the same time pointed out that epidemics of enteric fever were often caused by polluted drinking-water. The enteric bacillus was not known at that time, and the infection of the water was attributed to pollution by organic refuse, excrement, and such-like. If enteric fever bacilli find their way into a town's central water-service, and do not perish before the water is used for drinking purposes, a large number of inhabitants consuming the water may develop enteric fever in a short space of time, generally within a few days.

Infection will take place in a similar way, when a large number of persons partake of food specially infected with enteric bacilli. That is chiefly the case when a large number of people are supplied with milk from the same dairy, especially from a central dairy, even if the milk from one place only contain active enteric bacilli. Numerous instances, for decades past, in England, Germany, France, Sweden, etc., have been brought forward to prove that this can be, and not infrequently has been, the mode of spread. Here I only remind you of the enteric-fever epidemic of the summer of 1873, in Marylebone, St. John's Wood, and St. George's parish.

There is no question but that other articles of food can also, through enteric fever bacilli, be rendered dangerous to health. But it is only in exceptional cases that articles

of food other than milk will be consumed by a great number of persons simultaneously, for instance meat, oysters, etc. Therefore, transmission of infection by articles of food other than milk occurs only in isolated groups.

But it is always man who makes drinking-water and articles of food the vehicle of enteric fever bacilli. And it is man also who, in many cases, becomes the vehicle of infection, by transmitting enteric fever germs to his fellow-men, *i.e.*, he infects them. According to R. Koch, enteric bacillus has but a short existence in the soil anywhere outside the human body. The human body is almost without exception its habitation and place of multiplication. This theory of transmission by contact, which has been held since long times, taught by R. Virchow, and put on a scientific basis by the labours of Gendron, Tardieu, Budd and Gietl, was for a considerable time refuted by the adherents of the new school, or at least much disparaged. But at length, following the example of Robert Koch, this view has again found general acceptation. And we may with full authority assert that, without doubt, during an epidemic of enteric fever, a very large number of cases are due to transmission from man to man, directly or indirectly. This is true of all those cases when the persons attacked by enteric fever follow one another like the links of a chain, at a longer or shorter interval of time. By careful expert observation and estimation of the different cases, it will very often be possible to show the connection between the separate attacks. It is well known that the attempt to prove the presence of the enteric bacilli bacteriologically is not always successful in the case of pollution of water or articles of food. In many cases only an indication of them can be shown, as undoubtedly happened in the instance of the Gelsenkirchen epidemic of enteric fever, 1901-2, in Dr. Springfield's Report already mentioned on page 204.

After the above statements, it is hardly possible to deny the fact that uncleanliness of houses fosters the growth, increase, and spread of the enteric bacillus.

In the same way as there are latent cases of plague and cholera, so are there also cases of enteric fever which, on account of their mild course, are hardly recognisable by a doctor, much less by the persons attacked or by their friends. Such mild cases of enteric fever, which can often only be spoken of as suspected attacks, are, however, perfectly well able to introduce the bacillus to places far removed from the spot where it originated. This fact is

also of the greatest importance in combating the origin and spread of enteric fever. The attempt made to eradicate enteric fever in the Treves administrative area, which attempt was started nearly two years ago, has, according to R. Koch, established the fact that children are particularly frequently attacked.

What steps are now to be taken in order, with any success, to prevent the origin and spread of enteric fever, or indeed check it in the slightest degree? For centuries enteric fever cases have been to a certain extent looked upon as the barometer of the condition of public health—and rightly so. The very decrease of enteric fever in towns within and without Germany; for example, in Berlin, Frankfurt a./M., Wiesbaden, Bremen, etc., shows indisputably that the general sanitation in the circumstances, above-quoted for Berlin is able to effect a substantial decrease in the number of enteric-fever cases. And if we look back upon the past, we see that writers and investigators have repeatedly laid stress upon the fact that, in order to contend with enteric fever from the standpoint of public health, it is above all necessary to do away with those insanitary conditions which foster its spread.

Although the origin of enteric fever has now been discovered bacteriologically, and although it has beyond doubt been proved that the disease is spread by the bacillus, nevertheless we cannot restrict the measures taken against it to abolishing nuisances alone; but we must at the same time urge on general improvement of sanitation with all our might, in order to prevent the spread of the bacillus.

Nearly a century ago, before the bacillus was known, it was decreed (Prussian regulation of August 8th, 1835: measures against the spread of infectious diseases) that enteric patients should be isolated, and that their dejections should be disinfected, so that the disease could not be spread further by the discharges. And since it has been proved recently that enteric bacilli are often present in the urine in great amount, the urine must also be disinfected. Chloride of lime, carbolic acid, and milk of lime have long been considered to be the best disinfectants of the dejections and urine. Further, for the last twenty years steam has been of the greatest importance in the destruction of the germs in bedding, clothing, etc. New breadcrumb has been used with much success for rubbing the wall-papers of the sick-room. For washing the floors and other woodwork, green soap is a thoroughly good

disinfectant, which has only met with public recognition again in quite recent times. By all these means the bacilli are either destroyed or rendered harmless.

After the spread of the typhoid organism by personal contact had, thanks to the Gelsenkirchen epidemic of 1901-2, again been recognised even by the advocates of the almost exclusive drinking-water thesis, now Robert Koch showed that in combating enteric fever, the destruction of the bacilli must be aimed at in the first place, as in the case of cholera and malaria. Therefore, not only every actual case of enteric fever, but every suspected case must be reported, so that it can be determined as quickly as possible by bacteriological investigation, whether or no the person in question is harbouring enteric fever bacteria. In the event of its being proved in the affirmative, the patient should be isolated and treated with just as strict sanitary precautions as an ordinary genuine case of enteric. In every case the source of infection should be investigated. Moreover, persons suspected of having contracted the infection should be kept under observation, and if possible be subjected to bacteriological investigation. For bacteriological diagnosis, Koch recommends the Conradi and von Drygalski's method, which has answered well in the fight against enteric started about two years ago in several villages in the Hochwald, in Trier administrative area. This reaction, like the necessary bacterial examination, must be carried out by experienced bateriologists in a laboratory fitted up for the purpose. Medical men in practice and medical officials have neither the time nor the necessary skill for these delicate investigations. Robert Koch, in his Paper before mentioned, speaks of this method as an *experiment* to exterminate enteric fever: an experiment which had been successful in the Hochwald, near Treves, where the country had already remained free from the disease for a period of three months (p. 18).

Should this success be a lasting one, and should the attempt be made to render it lasting in other places where the disease is endemic, and where such laboratories are already in working order, bacteriology and hygiene would, in their warfare against infectious diseases, celebrate a further triumph hitherto undreamt-of for the well-being of mankind. But for the present it remains doubtful whether this advance against the enteric fever bacillus could effect such a radical change as to succeed in exterminating enteric fever in Prussia or the German Empire:

making it a "travelling disease" like cholera, recurring only periodically. It will only be possible to achieve this result when sanitation, not merely in Prussia or Germany, but also in all parts of the adjoining countries, has advanced so far that the enteric fever bacillus imported from without no longer finds a congenial soil. Whether further investigation on bacteriological lines will lead to the discovery that the enteric fever bacillus originates only by propagation; whether it can be evolved from other bacilli, or whether its toxines are the cause of the disease —all this must be left for the workers of the future to prove.

I come, therefore, to the final result that, in combating the origin and spread of enteric fever, general sanitation on the very best lines, according to the powers and progress of science, is to be continually insisted upon. Culture, also, with her daughters Freedom and Prosperity, according to Virchow's saying will in her further development substantially lighten the task of the authorities in their fight against this and other communicable diseases. It is on this basis that Robert Koch's proposed method can perform the task of routing out, or at least of limiting, the spread of enteric fever. For our knowledge of the immense importance of general sanitation we are, above all, indebted to those measures which for more than half-a-century have been employed in England, and which have there been crowned with such great success.

Vote of Thanks to Dr. Pistor.

The President: We have listened to an excellent and admirably-read Paper; and I think we must congratulate Dr. Pistor not only upon the matter he has so well put before us, but also on the success of his effort to address a foreign assembly at such length in a foreign tongue.

It has been a great pleasure to hear Dr. Pistor, and a great pleasure to welcome him once more, because he is by no means a stranger in this country. It is gratifying to us to find that Dr. Pistor, working along the lines of his experience in other countries, has arrived at almost the same results as we have in England. If we were going to discuss this Paper, there are two or three points which I think would well repay some time devoted to them. One in particular is the importance that Dr. Pistor—and no

„	„ Erfurt (12 Kreise)	3530,23
Prov. **Schlesw.-Holst.** (23 Kreise)		19002,42
Provinz **Hannover**		38509,80
Regierungs-Bezirk	Hannover (13 Kreise)	5716,44
„	„ Hildesheim (17 Kreise)	5350,18
„	„ Lüneburg (16 Kreise)	11343,80
„	„ Stade (14 Kreise)	6786,33
„	„ Osnabrück (11 Kreise)	6205,60
„	„ Aurich (7 Kreise)	3107,45
Provinz **Westfalen**		20209,23
Regierungs-Bezirk	Münster (11 Kreise)	7253,01
„	„ Minden (11 Kreise)	5259,87
„	„ Arnsberg (22 Kreise)	7696,35
Provinz **Hessen-Nassau**		15694,15
Regierungs-Bezirk	Kassel (24 Kreise)	10077,12
„	„ Wiesbaden (18 Krause)	5617,03
Rheinprovinz		26993,69
Regierungs-Bezirk	Koblenz (14 Kreise)	6204,84
„	„ Düsseldorf (25 Kreise)	5472,71
„	„ Köln (12 Kreise)	3977,05
„	„ Trier (13 Kreise)	7184,14
„	„ Aachen (11 Kreise)	4154,95
Hohenzollern		1142,26
Regierungs-Bezirk	Sigmaringen (4 Kreise)	
Preussischer Staat in Summa		348610,39

TEN YEARS, 1892 TO 1901.

1.52—1.94. 2.01.—2.62.

doubt with him other sanitarians—is inclined to attach to the slight and unrecognised cases. In many diseases, we regard these now as of the highest importance, not only because they form a link between more severe cases, but as supplying some sort of explanation of the mystery of what becomes of epidemic diseases in the times when they are not epidemic.

Then again, Dr. Pistor refers very forcibly to the idea of direct infection in typhoid fever. We in this country, as well as in Germany, are coming to recognise that as having more influence than at one time seemed likely to be attached to it.

I think the meaning of these results is this: that having recognised some of the main causes—water, milk, and so on—having dealt with these as they can be dealt with in many cases, other causes become more prominent when the total incidence is considered.

But I am tending to break the suggestion I myself made to the Society, and beginning what would be a discussion. I think the better course would be that I have suggested: that we simply receive Dr. Pistor's Paper as an address, and tender to him our grateful thanks for delivering it to us, and for coming so far to do so. I will, therefore, ask Dr. Murphy to propose a vote of thanks to Dr. Pistor.

Mr. SHIRLEY MURPHY: I beg to move that the grateful thanks of this Society be given to Dr. Pistor for his excellent and most able Paper.

I would like to add my own tribute to the value of this Paper. Coming as it does from an experienced administrator in Germany, it puts before us, in an excellent summary, the position of medical knowledge and medical views as to this disease in that country. I, too, was struck by the close similarity—the exact proportion of the different causes to which, in Dr. Pistor's mind, the distribution of this disease is to be attributed. We, too, in this country are coming to recognise that some things play a more important part than we had heretofore thought.

I read with great interest the Paper of Dr. Koch's to which Dr. Pistor has referred, in which he gives the results of his investigations in the neighbourhood of Treves, and in which he found numerous cases capable of spreading the disease, although not recognised clinically. He also found the method of Drygalsky and Conradi so useful, that I was tempted to suggest that it might be used at a certain institution with which I am connected, at which typhoid

had not only been endemic, but had shown abnormal prevalence for some time. The result is something that I am sure will astonish Dr. Pistor and everyone else: because from the very moment the laboratory was established and the bacteriologist engaged, we never had another case—the disease entirely disappeared. Of course, that is a result we cannot always anticipate; but I must say I do entirely believe that it is in looking after the cases which clinically do not present marked symptoms of the disease that we shall gain experience to guide us in getting rid of our remaining typhoid. Only to-day (although I do not come very much in touch with the actual investigation of disease), I learn particulars about half a dozen people who had been attacked by a disease which at first sight afforded no clear evidence of its nature; but after a time, I was told, two of the cases had presented marked symptoms of typhoid fever; three others had indefinite symptoms, and, read by the light of the other two, they were diagnosed as typhoid fever. That, I take it, is a very common experience; and that leads me to say that when we talk of the reduction of typhoid fever in this country, in Germany, and other parts where administrative measures have been applied, I do not believe it tells the whole story. I believe the reduction is really very much greater than is represented by the figures, and for this reason—that attention is now more frequently directed to the milder cases of the disease, the diagnosis is better, and had there not been a very great reduction indeed, these factors would have caused an apparent increase rather than a decrease; so that our efforts would seem to have been more productive of good even than the real figures would prove at the present time.

Again may I ask you to allow me to propose that the cordial thanks of this Society be given to Dr. Pistor.

Dr. NEWSHOLME: I feel it an honour to have the opportunity of seconding this cordial vote of thanks to Dr. Pistor for his admirable Paper; and one cannot help, in doing so, congratulating him upon the distinguished part he has taken in reducing the death-rate from enteric fever in Prussia from 6 to 2 per 10,000 of the population. That is a marvellous decrease, which is only equalled, I believe, by the corresponding decrease in our own country.

It is interesting to see how this question is approached from the German standpoint, and I think we can learn a great deal by the comparison. What particularly strikes

me is the importance that Dr. Pistor has attached to missed cases. I cannot help thinking with him that it is these cases which keep enteric fever alive in this and other countries, and I think the importance of notification of such cases will be increasingly recognised: because it is only by notification of suspected cases that one can hope effectively to eradicate this disease, and arrive at the stage which Dr. Pistor has very skilfully described of making enteric fever a mere "travelling disease," and not an endemic disease always in our midst.

Dr. Pistor has paid here a tribute to English sanitation of the past, and we, as representing the English school here to-night, may pay an even more enthusiastic tribute to the help which German science has given us in the prevention and diminution of infectious disease. When we remember what we owe to men like Koch in the prevention of tuberculosis, and also in the recognition of the bacillus of typhoid fever and other diseases, one cannot help feeling that German science has done as much, or almost as much, as English sanitation in bringing about the reduction of infectious disease during the last fifty years. One cannot help envying German sanitation in one respect, and that is in the centralisation of its organisation; also because it is not disturbed by the waves of public opinion so much as public health administration in this country is: in the matter of vaccination, for example. The results as regards smallpox in Germany afford a striking instance of what can be done when public health administration is allowed its own way.

Although the Germans attack the problem from a different standpoint to the English, it is remarkable that we have both of us arrived at the same result—the Germans more particularly from the bacteriological standpoint, and we more particularly from the nuisance removal standpoint. It appears, therefore, that it is of minor consequence at which point one breaks the links of the chain of infection, so long as the continuity of that chain is destroyed. The most efficient plan would be, not only to break the link in the chain, but also to destroy the opportunities of chain formation.

I am sure I am expressing the view of all the members of the Society, when I express the hope that this Paper will appear in our *Transactions*, with all the Tables attached to it, and with the Maps, which will make it even more instructive than we have found it this evening.

I beg very cordially to second the vote of thanks.

Dr. PISTOR: Mr. President and Gentlemen—I thank you with all my heart for the kindness with which you have heard my modest attempt to set forth the results of my experience of enteric fever. It was to me—as I have said in the beginning of my Paper—a very favourable and agreeable opportunity to come here and give you a short review of the behaviour of enteric fever in Prussia, and of the measures there taken against enteric fever. For the results of Dr. Koch's experiment we must, as I have said at the end of my Paper, wait. If he is successful it will be a good thing for mankind.

I thank you once more for all the kindness you have shown to me

TWO YEARS OF CHOLERA PREVALENCE IN THE TURKISH EMPIRE.

By FRANK G. CLEMOW, M.D. Edin., D.P.H. Camb., British Delegate to the Ottoman Board of Health; Foreign Secretary for Russia.

At the moment of writing, rather more than two years have passed since, in February, 1902, cholera was discovered among pilgrims journeying between Mecca and Medina. A great part of the Asiatic dominions of the Turkish Empire has since that time been overrun by the disease, and the total mortality, though not accurately—scarcely even approximately—known, has undoubtedly been very great. The behaviour of the epidemic in different parts of the country and during the varying seasons of these two years has offered many points of interest to the observer; and it will be the object of the present paper to place before the members of the Epidemiological Society the facts of this two years' prevalence of cholera, so far as they have been made known to the authorities in Constantinople.

A brief preliminary word may be said as to the extent of the Turkish dominions in Asia. They include Asia Minor, Turkish Armenia or Kurdistan, Syria, Mesopotamia, and a great part of the Arabian peninsula. With the exception of Asia Minor, all these regions have been visited by cholera during some portion of the two years in question, and each will be dealt with in detail in the course of this paper. A not less important chapter in the history of this epidemic has been the outbreak of the disease in Egypt, which formed indeed the link between the outbreak in Arabia and that in Syria. The course of the Egyptian epidemic must then be also briefly traced; but—if for reasons of space alone—at much shorter length than the course of the outbreaks in the other regions named.

It must be assumed, at any rate until the contrary has been proved, that the cholera infection was imported to the Hedjaz early in 1902, from one of the endemic centres of cholera in the East. During the fifteen preceding years the disease had appeared among the pilgrims to Mecca and Medina on four occasions only—in 1890, in 1891, in 1893,

and in 1894-5. For seven years, and during seven successive annual pilgrimages, the disease had, so far as is known, been entirely absent from the Hedjaz. That it had been present without its presence being known or recognised is a possibility so remote that it may be ignored. It will be shown immediately that the mode of its introduction to the Hedjaz was never determined; but if it be assumed—as it apparently must be—that India was directly or indirectly the source of the infection, then the behaviour of cholera there at and before the period in question is of interest.

The total cholera mortality in India in the two years 1898 and 1899 had been the lowest recorded for eighteen years. But in 1900 the deaths from this cause, which had numbered 152,703 in 1898, and 171,410 in 1899, suddenly rose to the enormous total of 797,222—"the highest total" (in the words of the Sanitary Commissioner with the Government of India) "of which we have any authentic record." In 1901, on the other hand, the deaths fell to 282,640. The year, therefore, immediately preceding the appearance of the disease in the Hedjaz was not a very bad cholera year in India, though, as just stated, the year before had been absolutely the worst cholera year on record.

The question arises whether the excessive activity of cholera in India in 1900 bore any relation whatever to the western extension of the disease, which only appeared to take place at the beginning of the second subsequent year, one year of comparative quiescence of the disease in India having elapsed in the interval. I have used the expression "appeared to take place" because, as a matter of fact, the outbreak of cholera in the Hedjaz in the early months of 1902 was not the first sign of a western extension of the disease from India. Already, in the autumn and early winter of 1899, an epidemic of some severity had been observed on the shores of the Shatt-ul-Arab, at Basra and elsewhere in the interior of Irak-Arabi, and even in Mesopotamia. This was followed, or rather accompanied, by a very severe epidemic of the same disease in the kingdom of Oman, in the south-eastern corner of Arabia. As little has hitherto been published concerning these two outbreaks of cholera, I propose to give in some detail what is known as to their origin and course, more particularly as it has been surmised by some that they may have served as connecting links between the outbreak in the Hedjaz and its original source in India.

The epidemic on the Shatt-ul-Arab—the river formed by the junction of the Tigris and Euphrates before their waters reach the Persian Gulf—began early in October, 1899. The first telegram recognising its existence was dated the 7th of that month. The disease had already at that date caused several deaths among the labourers employed in gathering in the date harvest. Every year, immense numbers of persons of both sexes are brought together from all parts of the interior to the date-groves on the shores of the Shatt-ul-Arab, where for several weeks they are accommodated in an exceedingly primitive manner, in what are called "chardaks": temporary structures which scarcely do more than afford them the barest shelter, and in which, apparently, the needs of hygiene, and even of decency, are entirely ignored. The extent of the mortality among these persons was never known accurately, but it was said to have been exceedingly high. At Basra —the most important town in this region—the epidemic was a comparatively mild one, and came to an end in the first week of December. The Bedawin tribes in the neighbourhood were considerably ravaged by the disease; and as many of them fled up into the interior, they were thought to have been the means of introducing the infection to the shores of the Tigris and Euphrates, and elsewhere.

It is impossible to determine the exact limits of the epidemic. Cases were officially reported from Nechoué, near the junction of the Euphrates and Tigris, from Amara on the Tigris, from Nasrieh, Souk-el-Shioukh, and other places on the Euphrates; and outbreaks of some severity occurred at Hay, among the tribes of Tasré, Sedjir, and Hadjil, in the Amara neighbourhood, and among the Hafira tribe, near Shatra-el-Amara. Bagdad seems to have escaped; and, although rumours were current that the disease had spread to Persian territory, the extent of the outbreak there was never known.

The cholera epidemic in Oman seems to have begun about the same time as that on the Shatt-ul-Arab. In this outbreak the infection was believed to have been derived from the opposite coast of Makran. Cholera had been present in Gwadar in August, 1899, and in four weeks had caused 368 deaths there. It was thought to have come to Gwadar direct from Karachi; and if (as is exceedingly probable) this was the case, no link is wanting between the Oman epidemic and the endemic homes of cholera in India. There is a constant and extensive traffic between the Makran coast and that of Oman, and the carriage of

infection from one to the other would be easy. The course of the epidemic in Oman was rather obscured by the presence of plague there at the same time, and in some of the earlier reports there was confusion between the two diseases. But it seems to be established that cholera caused a high mortality at Maskat, the capital; that it was even more severe and lasted longer at Mattra, the trade centre near Maskat; and that it ravaged a great part of the Oman coast, and spread far into the interior. At Tirir, near Sohar, on the coast of the Gulf of Oman, over 300 deaths were said to have occurred. At Sohar and at Sibb, on the same coast, the mortality was less. The northern coast, on the Persian Gulf, did not escape; and along the southern coast, on the Indian Ocean, the disease spread the entire length of the kingdom, as far as Dhofar, where the Oman coast joins that of the Hadramut. In the interior it was carried from *wadi* to *wadi* and from tribe to tribe, until it was believed that scarcely any part of the kingdom had escaped. The total mortality in this outbreak was estimated by one good authority at not less than 70,000 persons.

It has been surmised, as already remarked, that the outbreak of cholera in the Hedjaz in 1902 may have been caused, not by a direct importation of infection from India, but by carriage of the germ by pilgrims or travellers from Oman. But against this view must be set the facts that the epidemic in Oman was believed to have come to an end in the summer of 1900, or nearly two years before the disease appeared in Mecca; and that the earliest cases in the Hedjaz did not occur among pilgrims from Oman, or indeed from any part of Arabia. It is probable, therefore, that there was no direct connection between the two outbreaks.

Still another suggestion as to the source of the Hedjaz epidemic must be briefly considered. This is the suggestion that the infection was imported from Java, where cholera had been seriously epidemic in the summer and autumn of the year 1901. A very large number of pilgrims from the East Indies annually make the pilgrimage to the Moslem holy places, and the possibility that they had been the means of introducing the cholera infection to the Hedjaz could not be denied. But, on the other hand, there was nothing to show that they did do so. Six pilgrim vessels left Java in the months of September and October, 1901. On three of these ships no deaths from any cause occurred, either on the voyage or during the ship's stay at

Camaran or Jeddah. On a fourth vessel, three deaths occurred during the voyage, and three in the lazaret of Camaran. On a fifth there were six deaths on the voyage, and two in the lazaret; and finally, on the sixth, there were no less than seven deaths on the voyage, six more in the lazaret, and two on the run between Camaran and Jeddah, or seventeen deaths in all. This exceptional mortality caused much suspicion to attach to the vessel in question, and charges were freely made that the mortality on board her had been due to cholera, that it had been purposely hidden by the authorities, and that this was the real cause of the subsequent epidemic in the Hedjaz. A strict inquiry was made into all the facts connected with this incriminated vessel, with the result that the mortality on board her was shown to have been probably caused by ordinary diseases and not by cholera; that even if it had been caused by cholera, there was nothing to prove any connection between an epidemic in Mecca, which began in February, and the arrival of a steamer at Jeddah in the preceding October; that the earliest cases did not occur among Javanese pilgrims, and that, therefore, the origin of the outbreak must be sought elsewhere.

Whatever may have been its origin, the facts of the outbreak were briefly as follows. The first case of cholera in Mecca probably occurred on February 20th, 1902. Between that date and the 7th of March, 14 cases of suspicious character came to the knowledge of the Health Office there. It was only the fourteenth of these cases that was officially recognised as cholera, and reported as such to the central authorities in Constantinople. The first 13 cases had all occurred among Russian Cossack pilgrims, the fourteenth was a Takrouri. In the meantime, cholera had broken out among other pilgrims who had left Mecca for Medina. The earliest news received in Constantinople was in a telegram from Medina, dated March 4th, stating that the first caravan had arrived from Mecca on the evening of the 3rd, and that it had lost several of its number from cholera *en route*. The disease was said to have broken out in this caravan at a place called Kadimeh, near to Rabouk, about half-way between Mecca and Medina. As the journey from Kadimeh to Medina would occupy about five days, it must be supposed that the disease appeared in this caravan about the 26th of February.

To make this course of events quite clear, it should be stated that the majority of pilgrims from the south land at Jeddah, proceed from there to Mecca (a distance of only

65 miles), and after a short stay there travel northward to Medina. The journey between the two occupies about ten or twelve days. After a stay of only a day or two in Medina the caravans start back, in order to arrive in Mecca in time for the three days' fêtes of Kurban Bairam, with which the Haj comes to an end. Pilgrims from the north, on the other hand, usually land at Yambo, and visiting Medina first, proceed thence, separately or with the others, to Mecca; whence they generally travel to Jeddah, where they take boat to return to the north.

The nationality of the pilgrims first attacked on the road between Mecca and Medina was never stated; but the caravan consisted of pilgrims of Turkish, Bokharan, Javanese, and Takrouri nationality. Several successive caravans continued to arrive at Medina, and all told the same tale of severe suffering and mortality, but only after having reached Rabouk. This northern half of the road between the two holy cities seems to have been infected for some considerable time before, and more severely than, the southern half of the road. Thus the Mahmel-i-Sherif, or grand caravan with the holy carpet, left Medina on March 6th, and arrived at Mecca on the 16th, and stated that "as far as Rabouk the disease had caused a high mortality," but that "after that village the number of cases had gradually diminished." Other caravans had the same experience, but later the whole road seems to have become infected, and it was stated that "the inhabitants of the villages of Bir-bin-Hassani, Bir-Sheikh, Mastura, Rabouk, Kadimeh, Kouleis, and Asfan had paid heavy tributes to the cholera."

The fact that the earliest cases of cholera in Mecca —and therefore in the Hedjaz—occurred among Cossack pilgrims from Russia naturally raised the question whether these persons were not the means of introducing the infection from their homes in Russia; but there is reason to believe that Russia had been entirely free from all traces of cholera for some five or six years before the disease appeared in the Hedjaz. This view, therefore, finds no more to support it in the facts than the other views already mentioned, and it must be admitted that the origin of the Hedjaz epidemic has never been ascertained. All that is clear is that, once again, as on so many previous occasions, the great annual converging stream of thousands of persons from all parts of the Moslem world was the means of bringing cholera to the Hedjaz from some other place undetermined; and that it was thus responsible for the two

years' prevalence of the disease in the other parts of the Near East mentioned above.

While the pilgrimage of 1902 was thus seriously ravaged by cholera, it is satisfactory to note that that of 1903, and that of 1904 just terminated, have passed without any recurrence of the disease. Whether this is to be ascribed to the measures taken to prevent a recurrence, or to other causes, is not easy to say. That the measures now put in force each year to control the Haj and to limit the chances —still considerable—of its carrying infectious diseases, have a very real effect can scarcely be denied; nor can it be questioned that this very outbreak, two years ago, led to a marked improvement in the sanitation of Mecca itself, where the greatest concentration of pilgrims takes place. It would be tempting, did space permit, to describe in some detail the reforms that have been introduced there. Everything, indeed, connected with whatever aspect it may be of the Moslem Haj and its relation to disease is so full of interest, that not one but many papers might be written without exhausting the subject. It is, however, a subject strictly foreign to the scope of the present paper, which merely aims at setting forth the facts of the recent cholera prevalence; but, even at the risk of tiring the reader, there is one aspect of the Haj which deserves to be considered here. This is the question as to the number of pilgrims who annually flock to the Hedjaz.

It may be at once stated that the exact number of pilgrims in any Haj has never been known. A more or less exact knowledge of the numbers who arrive in the Hedjaz by sea can always be obtained; but these only form a portion, and a very varying portion, of the total number. Scarcely even an approximate estimate is possible of the numbers arriving by land. Still further confusion has been introduced by the practice so frequently followed of estimating the number of persons present at the final fêtes of Kurban Bairam, in the valley of Mina, and quoting the figure arrived at as the number of pilgrims to the Hedjaz. The inaccuracy of this method will be at once apparent when it is pointed out that the gathering of true pilgrims is enormously swelled by an immense crowd of persons — guides, camel-hirers, traders, inhabitants of Mecca itself, Bedawins from the desert around—none of whom deserve the name of pilgrim in any sense. It is practically certain, therefore, that the estimates often made in the past have been very considerably above the truth. The estimate therefore made in the present year (1904), on

good authority, that about 200,000 persons were present at Mina may be taken to indicate that some 100,000 pilgrims have reached the Hedjaz from elsewhere.

To return from this digression: the course of the outbreaks in Mecca and Medina varied exactly with the movements of the pilgrims. Thus, in Medina, when the return flow of pilgrims to Mecca set in, the mortality rapidly declined; and from a maximum of 86 deaths, officially reported on March 6th, the daily number of deaths fell rapidly, until on March 12th the deaths were numbered by units. Sporadic cases still occurred until the 18th of April, and in all, 348 cholera deaths were officially reported. This figure includes 68 deaths among the residents of the town, and 6 among the garrison. The distribution of these deaths by nationality is shown in Table I, and the daily returns of mortality in Medina are set forth in Table II.

In Mecca, the totals were less accurately known. For several days after the disease was recognised there, it continued to cause only sporadic cases; and it was not until the large mass of pilgrims were assembled for the fêtes of Kurban Bairam, which fell that year in the third week of March, that the mortality from cholera began to be serious. The total mortality here was never known, but it was undoubtedly very great. The daily returns are shown in Table III.

With the presence of cholera at both Mecca and Medina, it was inevitable that the two pilgrim ports of Jeddah and Yambo should sooner or later become infected. The first case in Jeddah was observed on March 10th. The patient was a pilgrim from Hadramut, and had arrived at Jeddah direct from Rabouk, the village on the road between Mecca and Medina, where, as stated above, cholera had first attacked the pilgrims. He died immediately after arriving at Jeddah. This death was soon followed by several others; and at the beginning of April, when the pilgrims were pouring down from Mecca to Jeddah in an unceasing stream, the cholera mortality reached its height. Outside the Mecca gate of Jeddah a sanitary station was established, and it was here that the larger number of deaths occurred. Every caravan arrived with its fresh contingent of sick, dying, and dead; and the total mortality was admittedly much higher than it appeared from the official returns.

Table IV shows the numbers of pilgrims of different nationality who passed through Jeddah for Mecca, and the numbers of deaths from cholera in each group after

their return from Mecca to Jeddah. The large number of Bokharan and other Russian pilgrims is explained by the fact that in 1902 Russia had removed the prohibition to her Moslem subjects, imposed in 1897, to make the pilgrimage to the Hedjaz.

At Yambo, cholera was first observed on March 30th, and the outbreak here only came to an end on June 8th. The total recorded deaths numbered 243; their distribution by nationality is shown in Table V. Before leaving the Hedjaz, it should be stated that small recurrences of cholera were observed at Medina in May and September, 1902. In the first recrudescence 15 deaths, and in the second 35 deaths, from the disease were reported to have occurred there.

A little more than a month after cholera appeared in the Hedjaz, it broke out in the province of the Yemen, in the south-western corner of Arabia. The exact date of its appearance here is uncertain. It is, however, certain that it was imported by the returning pilgrims, and that the district first affected was the neighbourhood of Abu Arish, in the northern portion of the province, and especially the large village or town of Sabia. A commission of three medical officers visited the district in May. They gathered that the earliest cases had occurred on April 14th; that the number of cases had already reached 300, and of deaths, 210; and that the numerous reassuring reports from the *kaimakam* of Sabia, to the effect that the outbreak was over, were erroneous, as they themselves saw several cases of the disease. Their efforts to arrange some measures of control were fruitless. The town council of Sabia stated officially that neither isolation nor disinfection could be applied, though they were ready to make use of ordinary measures of cleanliness, so far as they could make the people apply them. But a mass meeting of the inhabitants was held, and decided to prevent any further stay of the commission there, and used such threatening language that the commission was obliged to retreat to the coast. They were able to establish, however, that the disease was undoubtedly cholera, and that in addition to Sabia, the following thirteen villages were infected: Melcha, Meihalla, Shahir, Escha, Adaia, Muslia, Vassoli, Shukek, Djeinala, Eldzara, Selamatulia, Mulachap, and Djemiha. Cases of cholera also occurred in the port of Jizan. The extent and duration of this outbreak in the northern Yemen were never accurately known.

On June 9th, cholera appeared in the village of Salif, on

the Arabian coast, opposite the quarantine island of Camaran. Between that date and July 14th, 37 persons died from the disease out of a population of 1800. This small outbreak came to an end after the inhabitants had been supplied with distilled water from the Camaran lazaret, in place of the polluted well water they had been in the habit of drinking. At the time, no information was forthcoming as to the origin of the outbreaks in the Sabia district and in Salif, or as to any connection between the two. But some months later it was stated, on the authority of some of the local notables, of whom enquiry had been made, that the mode of introduction of the disease to Sabia, and its subsequent spread to Salif, had been clearly observed, and were as follows: A number of pilgrims returning from the Haj had landed secretly from a sambuk, or native boat, in a creek to the north of Jizan. One of the party—a woman—died of cholera at a place called Omel Hassap, and thence the disease spread to the other places already mentioned in the Sabia district. It also spread along the coast to Midi, and thence to Salif, where it was said to have been introduced by fishermen.

It is not easy to trace the exact course of the epidemic, but it seems to have spread from the northern Yemen northwards to the Assyr province, and southwards to the southern Yemen. In Assyr, the port of Confudah was infected in May and June, but apparently the mortality was not high, and the outbreak ceased on June 16th. An outbreak of far greater severity occurred at Mihail, and in a number of villages in the caza of Radjeb-el-Ma, in the interior of the Assyr province. It is impossible to give exact dates or statistics for the epidemics in these remote districts. In Mihail cholera was present at least as early as the month of July, and lasted until well on in October. Intermittent reports were received of the mortality it was causing there. Thus from August 14th to 28th, 47 deaths were known to have occurred. On October 18th, Mihail was visited by a sanitary official, and he found on enquiry that in the preceding five days as many as 360 cases of cholera and 150 deaths had occurred among three battalions of troops stationed there. There were 200 other cases then under treatment, and of these 65 died. This was probably a revival of the disease after a period of quiescence, as on September 28th it had been reported that no case of cholera had been seen there for twenty days. Outbreaks of the disease were also reported from Zobeideh and its neighbourhood, and from Attifé, among the Beni-Sheir tribe.

The area covered by these outbreaks of cholera in the interior of Assyr corresponds to some extent with the region where bubonic plague has been so long endemic.

On the Yemen coast, the parts of Loheia and Hodeida seem to have become infected some time in the month of September (1902). On the 29th September, it was reported by a military medical officer that at Mohr, in the neighbourhood of Loheia, 217 cholera deaths had occurred in the preceding 25 days. At Hodeida, the disease probably appeared on September 10th; the official figures for this port are given in Table VI. The mortality there in October was considerable, and probably much higher than would appear from the official figures. At the same time, the disease was reported to be raging at Morowa, Absia, and Halifa, all in the neighbourhood of Hodeida, but the exact degree of mortality was never known here. In Hodeida itself, the epidemic was said to have come to an end on November 2nd. An outbreak of great severity also occurred in November at Midi, a small harbour on the Yemen coast. Up to November 16th, as many as 150 fatal cases were said to have occurred among Turkish troops stationed there. So far as is known, cholera had disappeared from the whole of Arabia by the end of November, 1902, with the single exception of the port of Confudah, where sporadic cases of the disease were said to be still occurring at the beginning of February, 1903. Since that date there has been no trace of cholera in the Arabian peninsula.

In the meantime, during the summer of 1902, the cholera infection had passed all the barriers set up against it, and appeared in Egypt. The locality first affected was the village of Mousha, a few miles south of Assiout. The earliest case here was believed to have occurred on July 15th. Space will not permit of a detailed account of the epidemic in Egypt. The greater part of Lower Egypt, and a considerable portion of Upper Egypt, as least as far as Assouan, were invaded by the disease. By the end of the year 1902, when the outbreak was almost completely over, some 40,000 cases and 34,000 deaths (in round figures) had been reported in all. The manner in which the infection was introduced into Egypt was never clearly shown, but it scarcely admits of doubt that it was in some way imported from the Hedjaz by returning pilgrims. A strict quarantine was enforced at El-Tor on all such pilgrims, and a small number of cases of cholera (54 cases out of a total of 25,309 pilgrims interned in the lazaret) occurred there.

But none of the pilgrims from Mousha had been attacked by the disease at Tor.

Similar uncertainty exists as to the manner in which cholera was imported into Syria. The first news of its presence there was received at the end of September, 1902. It came in the form of a rumour that cholera had broken out in the *sanjak* of Hauran. The Hauran is a large district, south of Damascus, and through it runs the great pilgrim road from Damascus to the Hedjaz. This would naturally suggest the idea that returning pilgrims had brought the cholera infection here, as they in all probability did to Egypt. But at the end of September the flow of pilgrims for the 1902 season had long since ceased. Moreover, at about the same time, cases of the disease began to be reported from Gaza and its neighbourhood, on the Syrian coast. The most probable explanation, therefore, of the origin of the Syrian epidemic is that the infection was imported from Egypt, and not directly from the Hedjaz.

By the end of the year, cholera had spread northwards to Damascus, and eastwards to several villages in the district of Ajlun, to the east of the Jordan. Outbreaks of great severity occurred in Lydda, Tiberias, Gaza, Jaffa, and many other places. In Jerusalem, Jericho, and elsewhere, the epidemic was less severe. The total number of deaths officially reported from Syria up to the end of the year was 4,796. The larger number of these occurred in the *mutessarifate* of Jerusalem, or Palestine. This figure is unquestionably much below the truth. At Lydda, for example, the mortality, according to the official figures, was only 273, but a reliable authority estimated the deaths there up to the middle of November at 600, out of about 1000 cases, and similar discrepancies could be quoted for many other places. Table VII is a summary of the official returns up to the end of 1902, and Table VIII shows in detail the distribution of the cases and deaths reported in Table VII.

It may be noted in passing that during this early period of the epidemic the disease was spread to a considerable extent by the Italian and other labourers employed in constructing the new railway from Damascus to the Hedjaz. The disease at this time did not extend further north than Damascus. It steadily declined in December and January, and by the middle of February, 1903, it seemed to have disappeared altogether. But on March 18th it reappeared in Damascus, and from there spread in the following

summer months to a large number of towns and villages, not only in the vilayet of Syria proper, but also in the adjoining vilayet of Aleppo, to the north. Smaller outbreaks also occurred in the vilayet of Beirut, where, however, the only place of importance to suffer severely was the port of Tripoli. A summary of the official statistics of the 1903 epidemic in these three vilayets is given in Table IX. From this Table it will be seen that as many as 181 different towns and villages were invaded by the disease, and that the cases and deaths officially reported numbered respectively 7,823 and 5,483. These figures were unquestionably far below the truth.

The epidemic attained its maximum, as judged by the totals of cases and deaths reported, in the week ending August 10th. The figures returned for that week were 1,391 cases and 1,116 deaths. From that time there was a slow but steady decline in the outbreak in the Syrian vilayets; though, as will shortly be shown, the disease spread in the autumn to the Euphrates, and down that river to Mesopotamia and Irak-Arabi.

It will be impossible here to follow in detail the course of the epidemic from town to town, and from village to village. The disease followed, as it always has done, the main lines of communication. The high road from Damascus to Aleppo, *vid* Homs and Hama; the valley of the ancient Orontes river (for some distance coinciding with the high road just mentioned); the roads from Aleppo to Orfa and Biredjik; the valley of the Euphrates—these were some of the principal lines followed by the disease. Its spread by water was proved on many occasions. This was particularly the case in Damascus, where, it is worth noting, a novel method of dealing with a contaminated water supply was adopted. A stream flowing by one of the principal barracks was found to contain the cholera bacillus, at a time when cases of cholera had occurred among the troops in the barracks. The stream was temporarily dammed, its bed was covered with a layer of quicklime, and then the water was allowed to flow again. The local authorities claimed to have had good results from this proceeding.

The details of the epidemic in the Syrian vilayets, in the year 1903, are shown in Table X. The figures in this as in the other Tables are taken from the official statistics received by the Ottoman Sanitary Administration from their employés in the infected districts.

It is a noteworthy and fortunate fact that the Syrian

coast ports were but slightly affected. At Tripoli alone an outbreak of some severity occurred. Imported cases were seen at Beirut, and either isolated cases or small outbreaks of the disease at Caiffa, Alexandretta, Payas, and the island of Arvat, near to Tripoli. A single case also occurred on a ship in quarantine in the Clazomene lazaret, near Smyrna.

Cholera appeared in Biredjik, on the Euphrates, on August 16th, and six days later it was in Orfa (or Urfa), to the east. From here the infection spread north-eastwards to Diarbekir, and south-eastwards down the Euphrates to Kerbela and Basra. The valley of the Tigris was also invaded, and before the end of the year cholera had appeared in Bagdad and Mossoul. Table XI shows the official figures for the epidemic in the vilayets of Diarbekir, Mossoul, and Bagdad for the year 1903. It must, however, be added that the outbreak in Bagdad itself, though it probably began earlier, was not officially recognised until January, 1904; that the true mortality from cholera in the places named was very much higher than would appear from the official figures; and that the disease was known, from trustworthy sources, to have broken out in several places which did not appear in the official reports. The most serious outbreak was that in Kerbela, the city of burial for the Shiah sect of Moslems, and its neighbourhood.

Table XII is a recapitulation of Tables IX, X, and XI, and furnishes the official statistics for the cholera epidemic in all parts of the Turkish Empire in 1903. The total of 6,514 deaths should probably be doubled—or, as some even think, trebled—to represent the true mortality. By the end of the year 1903, it was believed that cholera was entirely absent from the vilayet of Syria proper; but on January 1st, 1904, it was reported that the disease had appeared in two places in the Hauran, and on the 3rd a "suspected" case was observed in Latakia. Apparently the infection has really died out in all parts of this vilayet since then. In the Aleppo vilayet, single sporadic cases were reported from Aleppo and Aintab in January, but since then this vilayet has also apparently been entirely free from the disease. With the opening of the new year, the scene of interest shifted to the regions watered by the Euphrates and Tigris, and the plains of Irak-Arabi, at the northern extremity of the Persian Gulf.

It was my original intention to confine this paper to a survey of the epidemic only up to the close of 1903. But it may fittingly be brought to an end by a brief *resumé* of

the behaviour of the disease in the first three months of the present year (1904).

At Bagdad, the first outbreak was short, and appeared to die out after January 7th. But exactly two months later it broke out afresh, and has continued to cause a small daily mortality throughout the month of March. At Basra, the epidemic began about February 6th, and it is still persisting at the moment of writing, with, if anything, a tendency to increase. Isolated cases have also occurred on steamers plying between Basra and Bagdad, and at places along the course of the river uniting the two. Rumours have also been heard that cholera had broken out at Meshed, in Persia, but there is reason to believe that these rumours are baseless. The danger, however, of the infection crossing the Turco-Persian frontier, and spreading up through Persia and Central Asiatic Russia to the Caspian, and so to Europe, is one which cannot be forgotten; as this, it will be recalled, was the course followed by the disease in 1892.

A few remarks may be made, in conclusion, on the general features of these two years of cholera prevalence in the various parts of the Turkish Empire. Whether due to the measures taken for its control, or to other conditions, such as a lack of excessive virulence or vitality in the virus originally imported, or unfavourable climatic or other conditions met with by it after importation, it is a fact that in no part of this Empire has the cholera outbreak been one of remarkable severity, or of long duration. In Arabia it was confined to a single Haj, and was fortunately extinct in the Hedjaz, though still present further south, when the pilgrims forming the next year's Haj began to assemble. In Egypt, the epidemic only lasted from July, 1902, to January, 1903; and though it covered a considerable area of the country, the total mortality was certainly less than in some previous epidemics, as, for example, that of 1883-1884. Similarly in Syria and Palestine, the spread of the disease was comparatively slow; it was strictly confined to the regions east of the eastern extremity of the Mediterranean, and (with the single exception of an imported case in the lazaret of Smyrna) did not extend to Asia Minor; and after one year of varying prevalence, cholera seems to have disappeared altogether from the Syrian vilayets. It is now present only in Mesopotamia and Irak-Arabi, and here also it has not shown itself in a very virulent form, nor has it, so far, exhibited a tendency to extend very widely. But when this has been said, it still remains the fact that, during

these two years of cholera prevalence, the total number of lives sacrificed to the disease has been a very considerable one. No means exist for making an exact estimate of their number; but it would probably not be above the truth to affirm that some 70,000 or 80,000 persons have succumbed to cholera in all parts of the Turkish Empire in the course of the last two years.

Constantinople, March 31st, 1904.

P.S.—Since writing the above, there has been a marked extension of cholera in Mesopotamia and Irak-Arabi, and the disease has already crossed the frontier and caused a considerable mortality in Persia. The second outbreak in Bagdad came to an end early in April, but the disease reappeared there a third time in the early days of June. Outbreaks of great severity have occurred at Basra, Nedjef, Hilleh, Shamieh, and elsewhere. The whole of the lower valleys of the Tigris and Euphrates are now more or less deeply infected, and the nomad tribes in the regions around are suffering to a considerable extent.

On Persian territory, cholera first appeared at Kermanshah early in April. The outbreak there has been one of great severity, but is now declining. The infection is said to have been largely spread by pilgrims returning from the Shiah city of Kerbela; it has caused outbreaks of varying severity at Khanikin and many other places on both sides of the frontier. Finally, it has recently been reported from the important centre of Kom, twenty-four hours from Teheran, and from the neighbourhood of Bender-Bushire, on the Persian Gulf. In the middle of May, cholera was also reported to be present in the Bahrein Islands, near the southern end of the Gulf, but no news has since been received of this outbreak.

June 24th, 1904.

TABLE I.—*Cholera Mortality in Medina in March and April*, 1902, *by Nationality.*

Nationality.	Men.	Women.	Children.	Total.
Indians	17	...	1	18
Bokharans	54	2	...	56
Afghans	3	...	...	3
Russians	11	2	...	13
Javanese	105	14	2	121
Persians	8	...	...	8
Soudanese	3	...	...	3
Algerians	5	...	...	5
Syrians	2	...	...	2
Anatoliotes	41	4	...	45
Inhabitants of Medina	47	10	11	68
Troops	6	...	...	6
Totals	302	32	14	348

TABLE II.—*Cholera Mortality in Medina in* 1902.
(Official Daily Returns.)

March	4th	...	40 deaths.	March	16th	...	3 deaths.
„	5th	...	77 „	„	17th	...	3 „
„	6th	...	86 „	„	18th	...	2 „
„	7th	...	41 „	„	19th	...	2 „
„	8th	...	17 „	„	20th	...	1 „
„	9th	...	12 „	„	21st	...	1 „
„	10th	...	13 „	„	24th	...	2 „
„	11th	...	14 „	April	14th	...	11 „
„	12th	...	4 „	„	15th	...	6 „
„	13th	...	6 „	„	18th	...	1 „
„	14th	...	3 „				
„	15th	...	3 „		Total	...	348 „

From April 18th to May 11th ... ... 0 deaths.
„ May 12th „ 20th ... ... 14 „
„ „ 21st „ 23rd ... ... 0 „
On May 24th ... ... 1 „

From May 25th to September 15th, no cases nor deaths.

From September 16th to October 9th, 35 deaths.

TABLE III.—*Cholera Mortality at Mecca in* 1902.

From February 20th to March 5th	... ...	12	deaths.
March 6th	... ...	1	„
„ 7th	... ...	2	„
„ 8th	... ...	3	„
„ 9th	... ...	4	„
„ 10th	... ...	6	„
„ 11th	... ...	4	„
„ 12th	... ...	13	„
„ 13th	... ...	11	„
„ 14th	... ...	14	„
„ 15th	... ...	2	„
„ 16th	... ...	21	„
„ 17th	... ...	9	„
„ 18th	... ...	15	„
„ 19th	... ...	10	„
„ 20th and 21st	...	31	„
„ 22nd*	... ...	220	„
March 23rd and 24th	...	247	deaths.
„ 25th	... ...	286	„
„ 26th	... ...	197	„
„ 27th	... ...	103	„
„ 28th	... ...	38	„
„ 29th	... ...	36	„
„ 30th	... ...	21	„
„ 31st	... ...	16	„
April 1st	... ..	21	„
„ 2nd	... ...	18	„
„ 3rd	... ...	11	„
„ 4th	... ...	7	„
„ 5th	... ...	4	„
„ 6th	... ...	3	„
„ 7th	... ...	2	„
„ 8th	... ...	1	„
Total	...	1389	deaths.

* Commencement of the Kurban Bairam fêtes. The figures here include the deaths at Mecca itself and in the Valley of Mina, where the Haj culminates in the sacrifice of thousands of sheep and in other ceremonies.

TABLE IV.

Nationality.	Numbers arriving at Jeddah for Mecca.	Cholera Deaths at Jeddah (after the Return from Mecca).
Indians	5,228	10
Afghans	432	
Bokharans	10,482	98
Russian subjects ..	1,480	
Javanese	6,481	11
Malays	2,976	
Persians	6,318	10
Hadramutis and Muscatis	301	5
Zanzibaris	7	—
Somalis	55	30
Sudanese	853	
Moroccans	528	47
Algerians and Tunisians	2,926	
Egyptians	5,486	3
Africans	9	—
Ottoman subjects :—		
From Hejaz and Yemen	2,803	43
„ Irak Arabi ...	444	
Syrians	1,072	
Tripolitans	743	
Anatoliotes	2,445	34
Rumeliotes	1,495	
Albanians	31	
From the Islands ...	21	
Soldiers	...	22
European	...	1
Total	52,616	314

TABLE V.—*Deaths from Cholera at Yambo in* 1902.

Nationality.	Men.	Women.	Total.
Bokharans	118	2	120
Persians	7	...	7
Takrouris	1	...	1
Moroccans	6	1	7
Algerians	41	3	44
Tripolitans	6	...	6
Ottomans	11	1	12
Egyptians	15	2	17
Syrians	3	...	3
Inhabitants of Yambo	8	5	13
Yemenlis	2	...	2
Bedawin	4	...	4
(?)	1	...	1
Afghans	2	..	2
Soldiers	4	...	4
Totals	229	14	243

TABLE VI.—*Cholera Mortality in Hodeida in* 1902.

On September 10th	4 cases ...	2 deaths.	
„ 11th	4 „ ...	4 „	
From „ 12th to Sept. 25th	0 „ ...	0 „	
„ „ 26th to „ 29th	14 „ ...	14 „	
On „ 30th	9 „ ...	8 „	
On October 1st	4 „ ...	2 „	
From „ 2nd to October 5th ...	25 „ ...	21 „	
„ „ 5th to „ 8th ...	32 „ ...	28 „	
„ „ 9th to „ 11th ..	23 „ ...	21 „	
„ „ 12th to „ 29th ...	(?) „ ..	26 „	

N.B.—The epidemic is said to have come to an end on November 2nd.

TABLE VII.—*Official Returns of the Cholera in Syria and Palestine in* 1902.

Name of Province and District.	Number of Towns in District.	Population of District.	Number of Towns Contaminated.	Total Cholera Deaths.
Mutessarifate of Jerusalem ...	328	341,638	42	3,620
Vilayet of Syria :—				
Damascus district ...	1,119	334,063	7	310
Ma'an „ ...	25	129,305	7	198
Hauran „ ...	258	137,312	10	113
Vilayet of Beirut :—				
Beirut district ...	323	171,663	2	59
Acre „	222	66,975	9	344
Balka „ ..	212	48,979	4	152
Totals	2,487	1,229,935	81	4,796

TABLE VIII.—*Detailed Returns of the Cholera Epidemic in Syria and Palestine in* 1902.

MUTESSARIFATE OF JERUSALEM.

Infected Places.	Population.	Date of Appearance of Epidemic.	Date of its Extinction.	Duration in Days.	Deaths.	Total Deaths by District.
Han-Yonnous .	3,000	Sept. ?	Oct. ?	?	221	
Batani Garbi...	600	Oct. 1 to 10	Oct. ?	?	55	
Gaza	30,000	Oct. 10	Nov. 23	44	937	
Lyddah ...	8,000	Oct. 12	Nov. 24	43	273	
Jaffa	65,659	Oct. 16	Nov. 29	74	268	
Deïr-ul-Bala ...	...	Before Oct. 17	Nov. 27	?	20	
Djebelia ...	6,000	Oct. 17	Nov. 28	42	77	
Beït-Djibbrine.	...	Oct. 19	Nov. 18	30	18	
Djemzou ...	...	Oct. 20	Nov. 11	22	83	
Beït-Nabale ...	...	Oct. 20	Nov. 13	24	50	
Sumeïl ...	...	Oct. 23	Nov. 24	1	1	
Yebna ...	1,000	Oct. 25	?	?	70	
Beït-Daras ...	...	Oct. 25	?	?	27	
Koufrana ...	1,000	Oct. 28	Nov. 22	25	184	
Beït-Djan ...	1,000	Oct. 29	Nov. 22	54	?	
Yahoudié ...	2,500	Oct. 30	Nov. 21	22	248	
Faloudja ...	2,000	?	?	?	69	
Nelia	200	?	?	?	25	
Ashdod ...	15,000	?	?	?	80	
Khsas ...	150	?	?	?	19	
Hamamé ...	500	?	?	?	29	
Tineh... ...	150	?	?	?	17	
Honj	200	?	?	?	30	
Koukab ...	1,200	?	Dec. ?	?	273	
Enab	?	?	Dec. ?	?	52	
Mejdeh ...	2,500	?	Dec. ?	?	80	
Hebron (Halil-ul-Rahman)	14,000	Nov. 2	Dec. 2	30	7	
Yazour ..	1,000	Nov. 6	Nov. 20	14	40	
Abou-Choucha	1,500	Nov. 7	Nov. 28	21	30	
Berieh ...	150	Nov. 7	Dec. 3	26	10	
Sakieh ...	250	Nov. 11	Nov. 22	11	40	
Sonafir ..	300	Nov. 11	Nov. ?	?	93	
Jerusalem ...	245,309	Nov. 14	Nov. 18	4	3	
Kezazé ...	?	Nov. 17	Dec. 11	25	7	
Nianeh ...	800	?	Dec. 20	?	20	
Safrieh ...	500	?	Dec. ?	?	40	
Mahmoudié ...	?	?	Dec. ?	?	3	
Yalo	?	?	Dec. ?	?	41	
Tournous-aya .	?	?	Dec. ?	?	17	
Yan	?	Dec. 2	Dec. 3	1	2	
Habareïn (near Jaffa)	?	Dec. 4	Jan. 4, 1903	31	33	
Jericho ...	400	Dec. 6	Dec. 16	10	28	
						3,620

Infected Places.	Population.	Date of Appearance of Epidemic.	Date of its Extinction.	Duration in Days.	Deaths.	Total Deaths by District.
VILAYET OF SYRIA.						
I.—District of Damascus.						
(Sandjak-Merkez).						
1,119 villages and 1,500 fixed tents=334,063 inhabitants.						
Hatem ...	.	Nov. 8	Nov. 25	17	11	
Erraya ...	...	Before Dec. 8	Dec. 9.	...	8	
Burkah ...	...	Jan. 1, 1903	Jan. 2, 1903	1	3	
Irbine ...	300	Jan. 1, 1903	Jan. 3, 1903	2	9	
Damascus ..	154,000	Jan. 5, 1903	Feb. 17	43	275	
Douma ...	8,365	Jan. 7, 1903	Feb. ?	...	?	
Djibbour ...	...	Jan. 7, 1903	Feb. ?	...	4	
						310
II.—District of Ma'an (Keruk).						
25 villages=129,305 inhabitants.						
Amman ..	...	Nov. 18	Nov. 21	33	67	
Arak Soudan. .	800	Nov. ?	Dec. ?	..	35	
Haresta ...	...	Nov. 29	Nov. 30	1	2	
Salt... ...	6,000	Dec. 1	Dec. 22	21	13	
Kafreïn ...	...	Dec. 13	Dec. 26	13	43	
Kufeyrat ...	...	Dec. 14	Dec. 22	8	8	
Arak el Menchie	...	Dec. ?	Dec. ?	...	30	
						198
III.—District of Hauran.						
258 villages=137,312 inhabitants.						
Hrer ...	800	Oct. 25	?	...	35	
Deïr-Tureïfé ...	...	Nov. 15	Dec. 5	20	29	
Wandel ...	...	Nov. 25	Nov. 26	...	4	
Sayara ...	...	Nov. 30	Dec. 10	10	3	
Ajlun ...	...	Dec. 1	Dec. 7	6	8	
Ragheb ...	...	Dec. 4	Dec. 12	8	25	
Husm ...	...	Dec. 5	Dec. ?	...	?	
Bakah ...	...	Dec. 14	Dec. 17	3	4	
Muzerib ...	...	Dec. ?	Jan. 2, 1903	...	3	
Kéfrendjé ...	...	Jan. 3, 1903	Jan. 4, 1903	...	2	
						113
VILAYET OF BEIRUT.						
I.—District of Beirut.						
(Merkez-Sandjak).						
323 villages=171,663 inhabitants.						
Ghazieh ...	...	Oct. 25	Dec. 5	41	35	
Bardja ...	...	Nov. 22	Dec. 6	14	24	
						59

Infected Places.	Population.	Date of Appearance of Epidemic.	Date of its Extinction.	Duration in Days.	Deaths.	Total Deaths by District.
II.—District of Acre.						
222 villages = 66,975 inhabitants.						
Tiberias ...	7,433	Oct. 24	Dec. 3	40	190	
Acre ...	10,400	Nov. 14	Nov. 29	15	12	
Mereké ...	...	Nov. 16	Nov. 19	3	10	
Melkieh ...	...	Nov. 18	Dec. 2	14	18	
Wadi-el-Bireh	...	Nov. 21	Nov. 22	...	4	
Meïss ...	...	Nov. 22	Dec. 5	13	4	
Saffed ...	10,000	Nov. 30	Dec. 5	5	2	
Abayat ...	...	Dec. 3	Dec. 4	...	8	
Kafr-el-Manda	...	Dec. 24	Jan. 4, 1903	11	96	
						344
III.—District of Balka (Nablous).						
212 villages = 48,979 inhabitants.						
Cheh-Moueness	400	Oct. 28	Dec. 14	47	80	
Tilkirn ...	...	Dec. 3	Dec. ?	..	?	
Oum-el-Fahm	...	Dec. 27	Dec. 31	2	2	
Nablous ...	24,830	Jan. 2, 1903	Jan. 24, 1903	22	70	152
Total known deaths from the beginning of the epidemic ...						4,796

TABLE IX.—*Statistics of the Cholera Epidemic of* 1903 *in the Vilayets of Beirut, Syria, and Aleppo.*

Vilayet or Circumscription.	District.	Number of Infected Places.	Cases.	Deaths.
Vilayet of Beirut ...	Sanjak of Beirut ...	3	49	26
	,, Acre ...	3	14	13
	,, Tripoli ...	2	396	317
	,, Lattakia ...	3	18	10
Vilayet of Syria ...	Sanjak of Damascus ...	50	2,935	2,260
	,, Hama ...	10	1,508	890
	,, Hauran ...	28	516	333
	,, Ma'an ...	4	43	38
Mutessarifate of Koudous	Sanjak of Jerusalem ...	3	110	63
Vilayet of Aleppo...	Sanjak of Aleppo ...	69	1,691	1,193
	,, Marash ...	1	5	2
	,, Orfa ...	5	538	338
Totals ...	... ...	181	7,823	5,483

TABLE X.—*Detailed Returns of the Cholera Epidemic in the Vilayets of Beirut, Syria, and Aleppo in* 1903.

VILAYET OF BEIRUT.

Infected Places.	Population.	Date of Appearance of Epidemic.	Date of its Extinction.	Duration in Days.	Cases.	Deaths.	Total of Deaths.
I.—DISTRICT OF BEIRUT (Sandjak-Merkez).							
323 Villages = 171,663 inhabitants.							
Ideydé ...	3,000	May 28	June 24	27	39	23	
Ghazieh ...	...	July 10	June ?	?	?	?	
Tel-Nehsan* ...	...	June 6	July 26	51	10	3	
Total ...	...	...	...	...	49	26	26
* One hour from Ideydé.							
II.—DISTRICT OF ACRE.							
222 villages = 66,975 inhabitants.							
Chaghour ...	...	July 4	Aug. 4	30	2	2	
Nazareth (Nasria)	8,000	Aug. 1	Aug. 2	1	1	1	
Caiffa	9,908	Oct. 25	Nov. 11	16	11	10	
Total ...	...	...	...	...	14	13	13
III.—DISTRICT OF TRIPOLI.							
567 villages = 96,109 inhabitants.							
Tripoli ...	30,110	July 22	Nov. 22	122	396	317	
Tartous* ...	...	Nov. 5	?	?	?	?	
Total ...	...	...	...	...	396	317	317
* Presence of cholera reported without details.							
IV.—DISTRICT OF LATTAKIA.							
1,240 villages = 149,830 inhabitants.							
Kadret ...	...	July 29	Aug. 2	4	2	2	
Aneyik ...	...	Nov. 7	Nov. 10	3	5	1	
Lattakia ...	22,000	Dec. 22	Dec. 30	8	11	7	
Total ...	...	...	...	...	18	10	10

VILAYET OF SYRIA.

I.—District of Damascus (Sandjak-Merkez).

Infected Places.	Population.	Date of Appearance of Epidemic.	Date of its Extinction.	Duration in Days.	Cases.	Deaths.	Total of Deaths.
Damascus .	154,000	March 18	Nov. 5	232	1339	1016	
Douma ...	8,365	May 13	Oct. 2	142	20	12	
Zebdani ...	...	May 24	Sept. 26	125	60	51	
Katana ...	2,150	May 24	June 25	32	45	29	
Beytaria ...	200	June 6	June 13	7	26	23	
Yabroud ...	...	June 13	Sept. 5	84	171	135	
Ibene	...	June 14	June 19	5	12	6	
Der-Ali* ..	250	June 17	July 5	19	108	93	
Kimaria ...	...	June 18	June 18	1	1	0	
Kessoné† ...	...	June 20	Aug. 25	66	46	22	
Hadar‡ ...	...	June 21	?	...	6	6	
Mó alaka ...	...	June 22	Aug. 19	58	5	2	
Der-Chemil ...	...	June 25	June 26	1	2	1	
Hidjani ...	...	June 30	July 23	23	90	28	
Tel-Hab§ ...	...	June ?	?	?	?	?	
Kerdjella‖ ...	...	July 1	Aug. 4	35	254	154	
Ma'azania ...	...	July 2	?	?	?	?	
Rahébé¶ ...	...	July ?	Aug. 10	?	350	310	
Larmié ...	...	July 4	Aug. 5	1	1	1	
Kouteipa ...	...	July 4	?	?	35	20	
Gabaguilé** ...	...	July 4	?	?	?	?	
Amra ...	...	July 4	July 5	1	1	1	
Tobar†† ...	...	July 4	?	?	?	?	
Medjani†† ...	...	July 5	?	?	?	?	
Cheghrania†† .	...	July 5	?	?	?	?	
Ou-el-Koussour †† .	...	July 5	?	?	?	?	
Doumer ...	...	July 5	July 10	5	7	7	
Michta ...	...	July 11	?	?	?	?	
Menyond ...	...	July 12	?	?	?	?	
Dormana ...	...	July 13	?	?	?	?	
Saran ...	...	July 15	July 15	1	1	0	
Haresta ...	...	July 18	?	?	14	14	
Ma'alaoula ...	...	July 18	July 28	10	6	6	

* South of Damascus.

† Three hours distance from Damascus.

‡ Three hours distance to north-east of Damascus.

§ A telegram of June 26th announced that in this village, in the district of Hamidié, ten deaths from cholera were occurring daily.

‖ Four hours south of Damascus.

¶ Total reported *en bloc* without detail.

** A telegram reported that after July 4th cholera was causing twelve deaths per day.

†† Near Der-Ali.

Infected Places.	Population.	Date of Appearance of Epidemic.	Date of its Extinction.	Duration in Days.	Cases.	Deaths.	Total of Deaths.
Ka'a*	...	July ?	Aug. 18	?	4	1	
Dera'ative ...	...	July 25	Aug. 25	31	53	34	
Djiroud ...	...	July 25	Aug. 30	35	40	35	
Ghaylania† ...	?	?	?	...	?	?	
Nehir ...	...	Aug. 1	Aug. 2	2	2	2	
Admir ...	...	Aug. 1	?	...	150	113	
Ontria ...	...	Aug. 3	Aug. 4	1	2	1	
Nebk	3,241	Aug. 4	Sept. 17	44	75	42	
Antebik ...	...	Aug. 5	Aug. 19	14	29	21	
Rayak ...	...	Aug. 14	Aug. 14	1	2	0	
Baalbek ...	5,000	Aug. 19	Sept. 22	34	88	55	
Bednaïl‡ ...	...	Aug. 19	Aug. 29	10	6	2	
Hadji-Pasha ...	...	Aug. 24	Aug. 24	1	3	1	
Horba ...	...	Aug. 24	Aug. 24	1	?	?	
Dareya ...	...	Sept. 7	?	?	21	11	
Sehneya ...	...	Oct. 8	?	?	6	3	
Manadanieh ...	...	Oct. 17	Oct. 19	2	2	2	
Total ...	...	...	...	...	2,935	2,260	2,260

* Eight hours distance from Baalbek.

† A telegram of July 28th reported that cholera was raging here with great intensity.

‡ District of Baalbek.

II.—District of Hama.

405 villages = 355,000 inhabitants.

Infected Places.	Population.	Date of Appearance of Epidemic.	Date of Extinction.	Duration in Days.	Cases.	Deaths.	Total of Deaths.
Hamidieh ...	5,000	June 8	July 11	33	3	2	
Hama ...	45,000	June 14	Oct. 26	134	606	348	
Boyer	...	June 17	?	?	10	9	
Aïn-el-Karoun	...	July 1	?	?	?	?	
Mervoud ...	...	July ?	?	2	22	11	
Homs ...	60,000	July 15	Oct. 2	79	853	515	
Selimieh ...	6,000	July 28	?	?	13	4	
Aneykié ...	...	Aug. 26	Aug. 27	1	1	1	
Yeni-Keuy ...	...	?	?	?	?	?	
Top-Boghaz ...	...	?	?	?	?	?	
Total ...	...	...	... ...	...	1,508	890	890

Infected Places.	Population.	Date of Appearance of Epidemic.	Date of Extinction.	Duration in Days.	Cases.	Deaths.	Total of Deaths.
III.—District of Hauran.							
258 villages = 137,312 inhabitants.							
Kara ...	...	June 7	Aug. 24	78	95	60	
Gabagheb ...	2,000	June 19	July 13	24	14	11	
Djebbal ...	1,000	June 17	July 5	19	14	7	
Mesiha ...	...	July 1	July 2	1	2	1	
Zahla ...	...	July 4	?	...	6	1	
Hadjira ...	...	July 5	?	...	?	?	
Kab-Elias* ...	...	July 15	Aug. 19	35	28	21	
Muzerib ...	...	July 16	Aug. 28	43	8	8	
Oknoum ...	...	July 22	July 23	2	2	2	
Mekiss ...	...	July 26	July 26	...	1	1	
Chedjara ...	...	July 28	Aug. 8	11	209	129	
Haouché-Hala ...	...	July 29	Aug. 3	6	3	3	
Merdjé ...	...	Aug. 1	?	...	?	?	
Hamah ...	...	Aug. 2	Aug. 2	...	1	1	
Kesireh ...	...	Aug. 4	Aug. 8	5	9	9	
Han-Donoum ...	...	Aug. 4	Aug. 4	...	1	1	
Yakousza ...	...	Aug. 6	Aug. 14	8	10	7	
Soueda ...	4,500	Aug. 20	Aug. 20	...	1	1	
Bekaa ...	...	Aug. 23	Sept. 7	16	44	21	
Farzel ...	...	Aug. 25	Aug. 25	...	1	1	
Ajlun ...	1,330	Aug. 28	Sept. 3	6	7	6	
Deraa ...	1,000	Aug. 29	Sept. 25	28	42	26	
Kefrendjé ...	...	Sept. 10	Sept. 12	2	2	2	
Selhad ...	4,000	Sept. 13	Sept. 13	...	1	...	
Basr-el-harir ...	1,500	Sept. 13	Oct. 12	30	12	12	
Cheïh-Miskine ...	...	Sept. 28	Sept. 29	...	2	1	
Nabe† ...	...	Dec. 30	?	...	?	?	
Inébé† ...	...	Dec. 30	?	...	1	1	
					516	333	333
IV.—District of Ma'an (Keruk).							
25 villages = 129,305 inhabitants.							
Kariak ...	...	July 13	June 17	...	3	3	
Aïn-et-Tineh ...	...	Aug. 1	Aug. 31	30	19	16	
Salt ...	7,700	Sept. 29	Sept. 30	...	2	2	
Keruk ...	2,000	Oct. 3	Oct. 28	26	19	17	
Total ...	...	...	...	...	43	38	38

* South-west of Mo'alaka.

† *Nahié* of Kuré.

Infected Places.	Population.	Date of Appearance of Epidemic.	Date of Extinction.	Duration in Days.	Cases.	Deaths.	Total of Deaths.
MUTESSARIFATE OF JERUSALEM.							
328 villages=341,638 inhabitants.							
Nahli ...	...	Aug. 2	Aug. 15	14	102	57	
Jericho ...	...	Aug. 12	Oct. 11	61	4	4	
Bethlehem ...	5,930	Oct. 15	Oct. 31	17	4	2	
Total ...	...	...	...	...	110	63	63
VILAYET OF ALEPPO.							
I.—District of Aleppo.							
2,750 villages=602,420 inhabitants.							
Tjoumassia ...	...	June 27	June 27	...	3	2	
Han-Cheroun	...	July 1	Aug. 4	35	8	6	
Djisri-Chough-our ...	...	July 19	Aug. 26	39	201	150	
Idlip ...	13,400	July 20	Oct. 6	79	50	41	
Kespan ...	...	July 21	July 21	...	1	1	
Kuk-Butchuk	...	July 21	July 21	...	1	1	
Maharah ...	...	July 31	Aug. 18	19	4	2	
Binieh ...	...	Aug. 1	Aug. 18	18	48	46	
Djisri-Hadid ...	...	Aug. 14	Aug. 21	8	5	3	
Maaret-el-Moursine ...	...	Aug. 15	Aug. 26	12	18	8	
Nableh ...	...	Aug. 17	Aug. 18	2	2	2	
Kafaroun ...	...	Aug. 18	Aug. 18	...	1	1	
Mirmiran ...	...	Aug. 20	Aug. 22	3	4	4	
Riha ...	13,000	Sept. 2	Sept. 8	5	9	8	
Alexandretta...	6,850	Sept. 4	Oct. 12	8	5	4	
Aleppo ...	127,149	Sept. 5	Dec. 3	90	429	400	
Tcheknedji ...	...	Sept. 10	Sept. 10	...	1	0	
Moustafa ...	...	Sept. 10	Sept. 12	3	3	3	
Asuakibé ...	...	Sept. 10	Sept. 27	28	22	12	
Hanfouek ...	...	Sept. 10	Sept. 14	4	3	3	
Hassan-Belou	...	Sept. 10	?	...	18	8	
Kara-Moustafa	...	Sept. 11	Sept. 12	...	3	2	
Haminab ...	...	Sept. 13	Sept. 13	...	1	0	
Harem ...	1,636	Sept. 13	Oct. 23	40	7	6	
Selkim ...	...	Sept. 13	Sept. 13	...	3	0	
Hazaz ...	...	Sept. 14	Oct. 13	30	111	58	
Bab... ...	5,722	Sept. 14	Sept. 21	8	21	10	
Tchayli ...	...	Sept. 14	Sept. 20	7	19	8	
Antioch ...	23,550	Sept. 15	Dec. 6	83	177	128	
Hassan-Keïn ..	...	Sept. 17	Sept. 18	2	2	2	
Hamza ...	...	Sept. 17	Sept. 29	13	34	14	
Salik-Bahri ...	...	Sept. 17	Sept. 17	...	1	1	
Kiffer-Messoud	...	Sept. 18	Sept. 18	...	1	1	
Tomfa ...	...	Sept. 19	Sept. 27	9	61	31	
Djaber ...	...	Sept. 19	Sept. 19	...	1	1	
Payas ...	...	Sept. 19	Sept. 20	2	5	5	

Infected Places.	Population.	Date of Appearance of Epidemic.	Date of Extinction.	Duration in Days.	Cases.	Deaths.	Total of Deaths.
Kizil-Hiss ...	...	Sept. 21	Sept. 21	...	1	0	
Koja ...	...	Sept. 21	Sept. 21	..	1	0	
Antass ...	...	Sept. 21	Sept. 21	...	1	1	
Ispadime ...	...	Sept. 23	Sept. 25	3	4	4	
Aïntab ...	43,150	Sept 23	Dec. 19	88	120	65	
Pasha-Babouk	..	Sept. 24	Sept. 27	4	16	6	
Hafiz ...	...	Sept. 25	Oct. 20	26	21	17	
Djiran-Tepé ...	.	Sept. 25	Oct. 27	3	3	3	
Yeni Sheïr ...	...	Sept. 26	Oct. 19	23	35	22	
Tel-Daout ...	...	Sept. 26	Oct. 16	21	5	4	
Ertah ...	...	Sept. 26	Oct. 6	11	11	8	
Pasha-Yol ...	...	Sept. 27	Sept. 28	...	2	2	
Tel-Talat Pasha	...	Sept. 27	Sept. 28	...	2	2	
Sarem ...	...	Sept. 30	Sept. 30	...	1	1	
Kadim ...	...	Oct. 7	Oct. 8	...	2	2	
Keuydjek ...	...	Oct. 8	Oct. 18	10	16	6	
Elif-oghlou ...	...	Oct. 8	Oct. 18	10	6	2	
Atkine ...	...	Oct. 10	Oct. 11	...	2	2	
Korkania ...	...	Oct. 19	Oct. 20	...	3	3	
Kara-Keuy ...	...	Oct. 20	Oct. 26	6	15	5	
Kara-Cheïh ...	..	Oct. 21	Oct. 26	5	16	5	
Ahdjéli ...	...	Oct. 24	Nov. 5	13	40	22	
Dolouki* ...	...	Oct. 25	Nov. 5	11	21	11	
Gneuk-Meydan	...	Oct. 25	Nov. 5	11	18	7	
Hassan-Moussa	...	Oct. 26	Oct. 28	3	3	3	
Bey-Keuy ...	...	Oct. 26	Oct. 27	2	1	0	
Kiliss-Ennu ...	...	Oct. 26	Nov. 5	11	9	5	
Yeni-Saïnt ...	...	Oct. 26	Oct. 30	4	4	4	
Djileïh ...	...	Oct. 27	Nov. 2	7	4	3	
Karata ..	...	Oct. 28	Nov. 2	6	8	4	
Kaaneyé ...	...	Oct. 29	Nov. 2	5	15	5	
Lostcha ...	...	Nov. 2	Nov. 2	...	1	0	
Hadjili ...	...	Nov. 25	Nov. 25	...	1	0	
Total ...	...	...	...	...	1,691	1,193	1,193

* Near Beylan.

II.—District of Marash.

552 villages=179,853 inhabitants.

Andérine ...	21,119	Aug. 21	Aug. 24	3	5	2	2

III.—District of Orfa.

1,239 villages=143,485 inhabitants.

Souroutch ...	1,500	Aug. 16	?	...	35	17	
Birédjik ...	10,162	Aug. 16	Dec. 1	108	252	123	
Orfa ...	55,000	Aug. 22	Nov. 20	91	218	180	
Mezrakeui ...	...	Sept. 9	Sept. 9	...	1	0	
Haran ...	31,625	Sept. 10	Sept. 17	8	32	18	
Total ...	...	...	...	...	538	338	
Total deaths in the three vilayets in 1903							5,483

TABLE XI.—*Returns of the Cholera Epidemic in the Vilayets of Diarbekir, Mossoul, and Bagdad in* 1903.

VILAYET OF DIARBEKIR.

Infected Places.	Population.	Date of Appearance of Epidemic.	Date of Extinction.	Duration in Days.	Cases.	Deaths.	Total of Deaths.
DISTRICT OF DIARBEKIR.							
1,056 villages = 143,923 inhabitants.							
Diarbekir ...	35,000	Nov. 21	Dec. 31	42	65	44	44
VILAYET OF MOSSOUL.							
DISTRICT OF MOSSOUL.							
811 villages = 159,680 inhabitants.							
Mossoul ...	61,000	Dec. 26	Dec. 31	...	1	1	1
MUTESSARIFATE OF ZOR.							
DISTRICT OF ZOR.							
158 villages = 45,000 inhabitants.							
Meyadine* ...	1,750	July ?	?	...	49	28	28
* Reported *en bloc* without details.							
VILAYET OF BAGDAD.							
I.—DISTRICT OF BAGDAD.							
60 villages = 400,000 inhabitants.							
Anah	3,000	Oct. 29	Dec. 3	35	526	281	
Djibbah ...	...	Dec. 7	Dec. 8	...	2	1	
Hitt	...	Dec. 13	Dec. 15	3	8	4	
Hadissa ...	...	Nov. 20	Dec. 6	6	18	8	
Total ...	...	...	...	...	554	294	294
II.—DISTRICT OF KERBELA.							
200,000 inhabitants.							
Kerbela ..	65,000	Dec. 9	Dec. 23	14	674	634	
Hindieh ..	2,000	Dec. 18	Dec. 19	2	2	2	
Musseyeb ...	...	Dec. 18	Dec. 29	11	33	28	
Total ...	...	...	...	...	709	664	664
Total deaths in the three vilayets in 1903							1,031

TABLE XII.—*Recapitulation of Tables IX, X, XI. Summary of the Cholera Returns from all parts of the Turkish Empire in* 1903.

Vilayet or Mutessarifate.	Districts.	Number of Infected Places.	Cases.	Deaths,
Vilayet of Beirut ...	Sanjak of Beirut ...	3	49	26
	,, Acre ...	3	14	13
	,, Tripoli ...	2	396	317
	,, Lattakia ...	3	18	10
Vilayet of Syria ...	Sanjak of Damascus ...	50	2,935	2,260
	,, Hama ...	10	1,508	890
	,, Hauran ...	28	516	333
	,, Ma'an ...	4	43	38
Mutessarifate of Koudous	Sanjak of Jerusalem ...	3	110	63
Vilayet of Aleppo...	Sanjak of Aleppo ...	69	1,691	1,193
	,, Marash ...	1	5	2
	,, Orfa ...	5	538	338
Vilayet of Diarbekir	Sanjak of Diarbekir ...	1	65	44
Vilayet of Mossoul	Sanjak of Mossoul ...	1	1	1
Mutessampote ofZor	Sanjak of Zor ...	?	49	28
Vilayet of Bagdad	Sanjak of Bagdad ...	4	554	294
	,, Kerbella ...	3	709	664
Totals ...	... ...	191	9,201	6,514

OBITUARY.

Sir John Simon, K.C.B.

The last of the little band of scientific spirits who founded our Society more than half a century ago has passed away: John Simon, one of the men of genius who made the reign of Queen Victoria illustrious, the first Chief Medical Officer of an English Public Health Department, died at the end of July, 1904, after a very long period of retirement from official life, at the advanced age of 87¾ years.

It was in 1876 that he left the then new offices at Whitehall, and the chair now occupied by the fifth Chief Medical Officer, viz., Mr. William Henry Power, C.B., F.R.S.

The time of Mr. Simon's resignation was one of great worry and inter-departmental friction, which must have seemed to him in strange contrast to that period of peace and honour passed at the old office over the way. It was there, at No. 8, Richmond Terrace, that the little group of Public Health official workers, all men distinguished for their scientific or administrative ability, used to meet together and discuss with their chief, to whom they were all loyally attached, the conduct of those epidemic inquiries and etiological investigations which made the work of the department so famous during or about the "'Sixties." It was there, too, that the administration of systematic Public Vaccination and the control of epidemic smallpox was practically founded and developed on a system which was in accordance with the best information and experience of the time. In the position of dignity and almost autocratic authority in which Simon, undecorated, but influential by right of his great talents and knowledge, was enabled (acting with statesmen such as Lord Sherbrooke and others with whom he was in most friendly relations) to direct inquiry and research, in a way that not only the nation but the world at large received immediate and immeasurable benefit therefrom.

As to the causes of Simon's retirement, and the fundamental principles of official administration which their discussion necessarily involves, we have no concern at present. The rights of the question were always clear to Sir John; and, as will be remembered, he relieved his mind at considerable length in his volume on English Sanitary

Institutions, published in 1890. But in this notice we must simply confine ourselves to a brief consideration of the life-work and position of the first Chief Medical Officer as a man of science and wisdom who, by virtue of his great qualities, was enabled to promote in a high degree the objects for which our Society was originally founded.

Sir John Simon, born in London on October 10, 1816, was of Anglo-French descent. He had great educational advantages, his boyhood being spent partly at a very good English school, partly with a German pastor at Hohensolms, in Rhenish Prussia, where he acquired a colloquial knowledge of German and formed enduring friendships, and partly with his French relations, with whom he used to speak the language as in the mother tongue. Then in the autumn of 1833 it was planned that he should commence the study of surgery, with a view to eventual practice as a consulting surgeon. For that purpose, according to the custom of the time, he had to be made, for six years, the apprentice of a London Hospital Surgeon; and his father decided that the very eminent person whose pupil he was to become should be Joseph Henry Green. This close relationship of teacher and apprentice was of inestimable advantage to Simon, and must have influenced his subsequent career in a profound degree. In a famous address which he gave to students before the Governors of St. Thomas's, fourteen years later, the pupil spoke feelingly of his old master, and of his deep obligation to his great intellect; referring to him as "one whom in the Councils of our profession and in the foremost van of European philosophy, stood conspicuous by wisdom, by virtue, by dignity, by eloquence."

In 1838, a year before the expiration of his hospital apprenticeship, Simon qualified at the College of Surgeons, in order to take the post of Demonstrator of Anatomy at King's College; and in 1839, after completing his sixth year of hospital pupilage, he began to be a housekeeper in London, and for fourteen years (1839 to 1853) was resident "in the then quiet of Wellington Street, leading to Waterloo Bridge—which in those days was not passed except with payment of toll," living for the first seven years in a little house on the east side of the street, "which had its back looking into the silence of Somerset House, and which, with downstair premises as deep as those upstairs were high, was singularly convenient for physiological purposes." This period comprised the chief part of his "supposed waiting time;" and now we find him not content with devoting part of his time to metaphysical reading

and the study of Oriental languages, by which he further increased the extraordinary wealth of culture which afterwards gave such interest and dignity to his speeches and writings, but also writing on medical education, and engaging in important initiative scientific work. In 1842 he wrote the article "Neck" for the *Cyclopædia of Anatomy and Physiology;* in the spring of 1844 he gained the first Astley-Cooper prize by a physiological essay on the Thymus Gland (which was published, with additions, in the following year), and had written for the Royal Society a paper on the "Comparative Anatomy of the Thyroid Gland," which gained him his Fellowship in 1845, when he was hardly twenty-nine years old. Two years later he furnished to the Royal Medico-Chirurgical Society his remarkable paper on "Sub-acute Inflammation of the Kidney."

It was by achievements of this high order that Sir John compelled the attention of the scientific world quite early in his career; and it is not surprising, therefore, to find this comparatively young man being invited in 1847 to take the first Chair of Pathology, and to become surgeon at his old and familiar home in St. Thomas's, where, with progressive changes of title, he remained an officer till his death, fifty-seven years later. It was Sir John's habit to refer modestly to his youthful papers, apologising "for what they no doubt contained of error," and speaking of himself as "learning to feel an interest in science." But let anyone carefully read his first lecture at St. Thomas's, delivered before a large meeting of the Governors and friends of the old school, who were present with his students, and he cannot fail to recognise the well-disciplined reasoning of the eminently practical man of science.

The lecture is devoted to the consideration of the condition of Pathology in his day, which he discusses under three heads: (1) Interpretation of Phenomena, (2) Doctrine of Cause, and (3) Principles of Classification. It is prefaced by quotation from the *Novum Organon*, and contains here and there rhetorical passages, which show how deeply the author was inspired by reverence for the teachings of Lord Bacon.

Some of these are worth preserving, if only as examples of Simon's unapproachable style:—

"For the pride and for the profit of mankind, Lord Bacon has lived; not vainly. Day by day, during the last two centuries, his mighty genius has been a living presence in men's minds; standing vividly there, as in the clear horizon of daybreak; pointing from the unillumined earth to the kindled sky; pointing from the chaos of undisciplined observation to the sublime possibilities of the inductive philosophy. In proportion as Science has conformed to the instructions

of him, her chief legislator, so has her dominion been extended and established; in proportion as she has turned from the course of legitimate induction, obsequious to traditions, or enamoured of idols of the fancy, in such measure have the foundations of her empire been shaken."

In the same strain, he concludes his powerful address with an inspiring exhortation to his pupils to study scientifically:—

"I would remind you that Pathology, on its own merits and independently of its application, is a subject of transcendent interest for the scientific inquirer. That the human mind, in proportion as it merits to be called human, can find no satisfaction or repose in any complexus of unexplained phenomena, all history bears witness. Amid even the most beautiful objects of sense, the human mind asserts its restless independence. In contemplating the sublimest of all natural spectacles, the midnight sky, 'with moving fires adorned innumerable,' who ever yet rested satisfied in the passive unspeculative perception of its vault, as 'thick inlaid with patines of bright gold'? Since first Chaldean shepherds gazed and trembled at its shifting phases, how it has riveted men's minds, as a mystery, is attested in the very names of the planets, is chronicled in the successive epochs of fire-worship, divination and astrology, and furnishes the explanation or excuse of a thousand genial superstitions. And now, when Kepler and Newton, with almost creative genius, have supplied the light for which centuries had yearned and thirsted; now when the harmony which poet-philosophers dreamed of has been realised to a degree which makes it almost a lesson of our childhood, how infinitely deeper is our sense of the beauty, and how more reverential our gaze; knowing as we do that those infinite orbs fulfil their appointed courses *in unity of law, with measurable precision of movement.*

"And, if this be true with respect to objects full of natural beauty and loveliness, how much more are all our moral interests enlisted to explore the sources of that which is physically evil and repugnant; to explain the possibility and significance of things which are awful to us, while unexplained, as apparent interruptions and catastrophes in the uniform and beneficent system of the universe! And how great the relief and the satisfaction to find that disease and degeneracy constitute no dark insoluble problem; to find that the laws of Life, in their own grand simplicity, include and account for all that, to the casual observer, seems hostile and capricious; to find nothing really abnormal; nothing in part or member foreign to the conservative intention of the whole; every contingency of disease included within those conditions which permit the possibility of health."

The man of genius who thus spoke had indeed already learnt "to feel a deep interest in science," which in after-years was destined to influence in a profound degree the welfare of his countrymen and humanity generally.

The official career which gave him such great opportunities for usefulness began with his appointment to the City of London health officership in 1848, which he held for five years. It was continued, as we have seen, in a higher sphere, till his retirement from official life in 1876. For about twenty years, with the enormous prestige he had

acquired, he must be said to have held a unique position, more influential than that of many a Minister of State. That he used it most wisely is universally recognised. It is unfortunate that he did not use it longer, as he, no doubt, might have done, if he had been prepared to accept the annoyance and provocations which were the common lot of officers in our Department, without resort to that extraordinary but dangerous power of sarcasm which he had cultivated when at the zenith of his fame, and during his close association with his ministerial chief and ardently-admired friend, the celebrated Mr. Robert Lowe, afterwards Lord Sherbrooke.

The splendid progress of Preventive Medicine made in Simon's time, and continued since by his able and distinguished successors has, as we all know, been due to strict adherence to scientific principles in the pathological and epidemiological investigations carried through by the Medical Department. It would be invidious to attempt to separate Simon's work from that of the brilliant colleagues with whom he was intimately associated during the best years of his life. The power of generalisation and other great qualities of mind, natural and acquired, which characterised the chief Medical Officer led the professional public to associate that epoch-making work too exclusively with his personality. It would be more correct to attach the great honour of affording rational explanation of epidemic occurrences, and adding to our knowledge of the intimate pathology of contagion to the Medical Department as a whole, as Sir John would no doubt have wished. The chief, as is the case now, never himself engaged in close epidemiological inquiry, holding himself in the high position of a judge on the work of others. This is, generally speaking, as it should be. But, unlike some of his successors in office, Simon had hardly ever himself carried out any of these laborious field observations and house-to-house inquiries, and consequently never quite appreciated what they involved, as shown by his inability to estimate the time which their conduct required. It is perhaps owing to this absence of first-hand observation that his judgment or generalisation may have shown a much greater appreciation of what the inquiries he directed disclosed than what they did *not* disclose. Still, from the point of view of practical epidemiology this is perhaps not to be regretted: for, to take one example alone, the doctrine that Typhoid is mainly a waterborne disease—if it did no more, supplied for many years "a good working hypothesis," as it was termed long ago by that

most sagacious physician, Sir William Gull. Or, to take another example from the experience of the last half-century in matters where science and administration are indissolubly connected, viz: the measures best suited to the control of epidemic small-pox. On vaccination no one could have written more powerfully than did Simon half a century ago; but on the technique of vaccination, and many of the administrative details and difficulties connected therewith, he was not so strong. To the last he retained his great interest in vaccination, but he did not fully comprehend the nature and extent of improvements effected in the performance of that beneficent operation. Nevertheless, his intellect being always keen, he grasped the bearing of modern sanitary organisation on the subject, and the promise it afforded by means of notification and isolation, etc., of material assistance in suppressing outbreaks of the dreaded epidemic disease—small-pox. But here again the experience of the physician and immediately practical administrator was essentially wanting to enable him fully to comprehend the difficulties connected with diagnosis, interference with "liberty" (so called), hospital establishment and administration: all of which further complicate the problem of epidemic control. Still, in the main, we can derive help and guidance by watching the movements of a master-mind, in considering an intricate question of administration in which our Society has been profoundly interested throughout its existence.

It is, indeed, as a master-mind that we must regard Sir John Simon, and we must think of him as one who, at a most critical period of the development of preventive medicine, did such truly great work in successfully introducing scientific principles into the details of sanitary administration.

Regarded from this point of view, the value of his labours was insufficiently understood or rewarded. For example, by applying the principles which he established to the subject of quarantine abolition, and the prevention of foreign epidemics by rational methods, he might be said to have earned the gratitude of the nation, and the reward that would have been readily accorded to any great lawyer or statesman for public services of a similar or analogous kind.

It is, mainly, to the fundamental nature of Simon's life-work that we desire to call the attention of our members in this brief and inadequate notice.

The Ordinary Meetings of the Society are held at

11, CHANDOS STREET, CAVENDISH SQUARE,

ON THE THIRD FRIDAY OF EACH MONTH FROM NOVEMBER TO MAY INCLUSIVE, UNLESS OTHERWISE ANNOUNCED.

THE CHAIR IS TAKEN AT HALF-PAST EIGHT O'CLOCK P.M. PRECISELY.

Subscriptions may be paid to the Treasurer. The Annual Subscription of Ordinary Members is One Guinea.

Ordinary Members are entitled to all privileges of membership, and to receive a copy of all the Society's publications issued during the year for which their subscriptions are paid. But Ordinary Members, resident beyond the limits of the Metropolitan Postal District, and having paid one or more annual subscriptions, may, on expressing to the Hon. Treasurer a wish to that effect, be distinguished as Non-Resident Members.

Non-Resident Members are liable to an annual payment of half-a-guinea. They have the privilege of attending meetings, and are entitled to receive the publications of the Society issued during the year for which their subscription is paid; but they are not entitled to vote at meetinys of the Society nor to hold office in it.

The Rules and Regulations, etc., of the Society, with an Exposition of its Objects, may be obtained from the Secretaries.

The Transactions for the Sessions 1859-60, 1866-67, *and* 1867-68 *are out of print. With these exceptions, those for the Sessions from* 1860-61 *to the last Session inclusive, may be obtained from the publishers,* Messrs. WILLIAMS AND NORGATE, 14, HENRIETTA STREET, COVENT GARDEN, LONDON, W.C. *Prior to* 1859-60, *the Transactions were published in the* "SANITARY REVIEW." *The issue of a New Series of the Transactions was begun in* 1882.